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A PRACTICAL TREATISE

ON

URINARY AND RENAL DISEASES.

A

PRACTICAL TREATISE

ON

URINARY AND RENAL DISEASES

INCLUDING

URINARY DEPOSITS.

ILLUSTRATED BY

NUMEROUS CASES AND ENGRAVINGS.

By WILLIAM ROBERTS, M.D.,
FELLOW OF THE ROYAL COLLEGE OF PHYSICIANS, LONDON; PHYSICIAN TO THE
MANCHESTER ROYAL INFIRMARY; LECTURER ON MEDICINE IN THE
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LONDON:
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PREFACE.

THE design of the present work is to give an account of the organic diseases of the kidney, and of those diseases and disorders of which the chief characteristic is some alteration of the urine.

The work naturally falls into three parts.

The first part—which may be regarded as introductory to the other two—is devoted to the physical and chemical properties of the urine, and to the various alterations which it undergoes under different circumstances of health and disease, in so far—and only in so far—as they seem to have a practical bearing. The methods of examining the urine for clinical purposes are explained; and the significance of the diverse changes experienced by it pointed out. The naked-eye and microscopical appearances of urinary deposits are described and figured, together with those of the extraneous matters which accidentally find their way into the urine.

Of the vast array of researches on the composition of the urine, and the rate of excretion of its several ingredients, accumulated in recent times, it has been found impracticable to give even an abstract, without greatly exceeding the limits of practical utility. It has seemed to the author more convenient to consign these purely chemical and physiological materials to separate treatises, in the manner adopted by Neubauer and Vogel and Dr. Parkes,—at least provisionally, that is, until such time as they can be shown to possess some clinical value. Further, these subjects are treated so amply in the works (in addition to those of the authors just mentioned) of Beale, Thudichum, and Hassall, that the omission of them has caused the author little regret. It is hoped, however, that nothing has been omitted a knowledge of which possesses any interest for the actual practice of medicine.

The second part treats of a group of affections which may be designated briefly as "urinary diseases," viz. diabetes insipidus, diabetes mellitus, gravel and calculus, and chylous urine. In his description of these diseases (with the exception of gravel and calculus), the author has endeavoured to present an analysis of all the facts hitherto published in relation to them, together with those which have fallen under his own notice. In the chapter on gravel and calculus, prominence has been given to the medical treatment, and especially to the author's own researches in this direction.

The organic diseases of the kidney form the subject of the third and largest part of the work. The present moment is not favourable for a lucid description of Bright's disease and its allies. Not only is there a wide divergence of opinion as to the clinical grouping of the cases, but the researches of the last three years indicate a necessity for a complete revision of our previous notions regarding the minute anatomy and the functions of the kidneys. The observations of Henle, and of the numerous inquirers who have followed him, have shown that the course and structure of the uriniferous tubes are far less simple than has been hitherto supposed; and the experiments of Oppler, Perls, and especially of Zalesky, challenge the very basis of the current opinion of the functions of the kidneys, and send us back to the older view, that the special ingredients of the urine—urea and uric acid—are actually formed by the kidneys, and not merely separated by them from the blood. A brief review of these researches is given in a note at p. 297, and at p. 359.

The less frequent affections of the kidney—hydronephrosis, cystic degeneration, cancer, tubercle, parasites, malpositions and malformations—are treated analytically, and at considerable length. The extreme poverty of the existing English systematic works on these subjects seemed to demand this compensation.

The author has not neglected any source of information within his reach. He is especially indebted to the works of Prout, Bright, Christison, Frerichs, Johnson, and Basham, and to the more comprehensive treatises of Rayer, Rosenstein, and Julius Vogel. A large quantity of material has also been collected from various English and Continental serials, and especially from the valuable issues of the London Pathological Society. Finally, the author is under a special debt of gratitude to his friend Mr. Thomas Windsor, whose labours during the last ten years, in connection with the library of the Manchester Medical Society, have alone rendered it possible to write the present work in this city.

To prevent the multiplication of foot-notes, the principal references have been placed at the heads of the several chapters.

MANCHESTER, 89, MOSLEY STREET,
October, 1865.

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PART I.

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CHAPTER I.

INTRODUCTORY.

I.—SUMMARY OF THE PROPERTIES AND COMPOSITION OF THE URINE: ITS PHYSIOLOGICAL AND PATHOLOGICAL VARIATIONS.

HEALTHY urine is a clear, watery, amber-coloured, saline solution, generally acid, with a specific gravity of about 1020. It contains a large quantity of *urea;* and smaller quantities of *uric acid*, *hippuric acid*, *creatine*, and *creatinine*. In addition to these, which are its characteristic constituents, the urine contains certain saline substances, namely, *chlorides*, *phosphates*, and *sulphates*, of which the bases are *soda*, *potash*, *lime*, and *magnesia;* also minute quantities of *oxalic* and *lactic* acids, *ammonia, pigment* and other substances which are classed under the head of *extractive matters.*

All these substances pre-exist in the blood, and are simply separated therefrom by the secerning action of the kidneys.

The average proportions of the chief constituents of the urine may be judged of by the following table, which has been constructed from a large number of the best analyses :—

	Water	954·81
	Solid matters	45·19
	Urea	21·57
	Uric acid	0·36
Extractives	Creatine, creatinine Ammonia, hippuric acid Xanthine, hypoxanthine Sarcine, pigment, unoxidized sulphur and phosphorus, mucus, &c., &c.	6·53
Fixed salts.	Chlorine	4·57
	Sulphuric acid	1·31
	Phosphoric acid	2·09
	Potash	1·40
	Soda	7·19
	Lime	0·11
	Magnesia	0·12

The composition and physical properties of the urine may undergo alterations from *physiological* and from *pathological* causes.

Physiological alterations.—The physical properties of the urine, and the relative proportion of its ingredients, vary greatly under the different conditions of healthy existence. Exercise, rest, the quantity and quality of the food and drink, digestion, fasting, sleep, the quantity of the cutaneous transpiration, atmospheric states, &c., react on the urine ; and are, so to speak, reflected in its composition.

Some of the urinary constituents are derived, wholly or in part, directly from the food. This is especially the case with the saline or mineral matters, and the water. When the diet is especially rich, or especially poor, in any of these, their relative proportions in the urine rise or fall correspondingly.

Again, certain constituents (especially water) have other ways of passing out of the body than the kidneys, namely, by the skin, the lungs, the intestines; and if these show any unusual activity the composition of the urine is necessarily affected. The greatest constancy of proportion is exhibited by the organic (nitrogenised) constituents—urea and uric acid, &c.—which are derived from the disintegration of the tissues; but even these oscillate not a little with the quantity and quality of the food, and with exercise or rest of the body. The reaction, which influences so importantly the physical properties of the urine, and its capacity for holding in solution certain ingredients which otherwise tend to be precipitated, is greatly affected by the digestion of food, and may be changed thereby from acid to alkaline during several hours in the day.

Pathological alterations may be distinguished into *general* and *special*. It is desirable to indicate these separately; though practically they frequently merge into each other.

General pathological alterations are those which depend on some general bodily disorder, such as fever, rapid waste of the tissues, anæmia, &c. Alterations of this class, although of great interest for the elucidation of general pathological doctrines, have very little symptomatic value; and it has not been shown that a particular knowledge of them in an individual case of disease, is capable of furnishing any information on diagnosis, prognosis, or treatment, which may not be obtained more easily and accurately by other means, namely, by physical examination of the organs, temperature-measurements, weighing the patient, &c.

Special pathological changes are:—(*a*) those in which some new and unnatural ingredient is mixed with the urine—such as albumen, sugar, fat, cystine, blood, pus, fibrine, epithelial cells, spermatozoa, &c.; (*b*) those in which some constituent is present in such unnatural proportion that the circumstance forms a leading feature of some particular disease—as the excessive quantity of water in diabetes, the excessive diminution of urea in Bright's disease, &c.; (*c*) those in which some constituent is in an unnatural physical condition—thereby producing or indicating a particular morbid state—as in the occurrence of uric acid, oxalate of lime and earthy phosphates as urinary deposits or calculous concretions.

In the present work, physiological and general pathological changes of the urine are only considered in so far as they possess some practical interest. The special pathological changes, on the other hand, are considered at length.

II.—METHODS OF EXAMINING THE URINE—APPARATUS REQUIRED.

An examination or analysis of the urine for clinical purposes is much more restricted in its objects than one which is designed for original investigations.

The object of the former is to ascertain those points, a knowledge of which, in a particular case, is known from previous experience to throw a light on the nature, course, diagnosis, prognosis, or treatment of the disease. The object of the latter is to obtain new and additional indications in the same directions; it embraces every conceivable information, and is consequently indefinitely elaborate.

The subjoined scheme is of the former kind, and is sufficiently simple to be within reach of every practitioner. It requires only an elementary knowledge of chemistry, and answers nearly all the requirements of actual practice.

The points requiring to be noted in an examination of the urine are—

1. The general appearance and colour; clearness or turbidity; presence or absence of deposit, and of extraneous impurities.
2. Odour.
3. Reaction.
4. Specific gravity.
5. Presence or absence of albumen: if present, an approximative estimate of its quantity.
6. Presence or absence of sugar: if present, an estimate of its quantity.
7. An estimate of the total quantity of urine in twenty-four hours.

If there be a deposit, note—

8. Its aggregation and colour: whether amorphous or crystalline, light or heavy; manner of subsidence or precipitation.
9. Solubility or insolubility by heat; solubility in nitric acid, in acetic acid, in liquor potassæ; insolubility in both acids and alkalies.
10. By the microscope:—absence or presence of crystals, their appearance and form; of epithelial cells—renal or extra-renal; of blood disks; pus globules; spermatozoa; fibrinous cylinders; confervoid vegetations, &c.

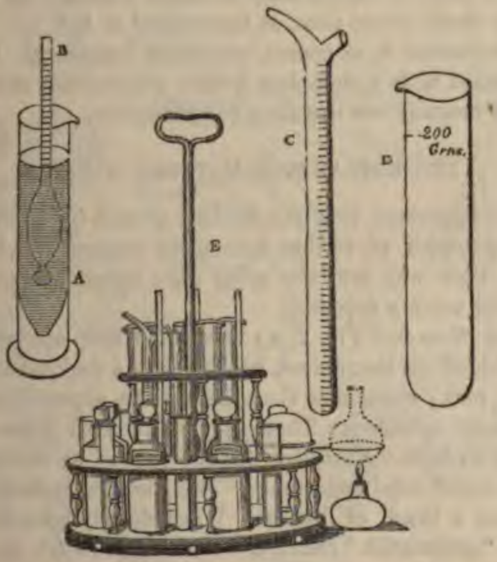

Fig. 1. Apparatus for urine-testing. A. Urine-glass—depth, 5½ inches; diameter, 1¼ inch. B. Urinometer. C. Burette, graduated in grains. D. 200-grain measure. E. Stand of urine-tests.

The apparatus required consists of—

1. Three or four urine-glasses. Fig. 1, A.
2. Litmus paper.
3. Urinometer. B.
4. Half-a-dozen test-tubes.

5. Spirit-lamp.
6. Nitric acid.
7. Acetic acid.
8. Liquor potassæ.
9. Liq. Ammon. fort.
10. Drop-tubes and stirring rods.

For Sugar Testing.

11. Prepared copper solution.
12. Graduated burette. C.
13. Two-hundred-grain measure. D.
14. Six-ounce graduated measure.
15. Small flask.

These may be conveniently arranged together for use on a circular stand of two tiers, as represented at E.*

A microscope is, of course, essentially necessary. It should be provided with a first-class $\frac{1}{4}$-inch object-glass, and an eye-piece to magnify not less than 240 diameters.

III.—EXTRANEOUS MATTERS IN URINE.

It is important that the student should be familiar with the appearance of certain extraneous matters which are apt to find their way into the urine after emission, and be mistaken for urinary deposits.

Cotton fibres (see Fig. 2, *a*.) have a flat limp appearance, are often folded on themselves, usually with a darker-looking medullary part; sometimes they present the appearance of narrow glassy cylinders. They vary in breadth from $\frac{1}{3000}$ to $\frac{1}{1000}$ of an inch. *Flax fibres* (*b*) are jointed at intervals, and have a round, solid appearance. Their broken ends are usually torn into a brush of fibrillæ. When sharply bent they break with a "green-stick" fracture. *Woollen hairs* (*c*) present the appearance of hard cylinders, with fine transverse markings and slight serrations along their margins. From their elongated form and somewhat similar diameters these three objects are liable to be mistaken for casts of the uriniferous tubes.

---

* This stand was constructed for me by Mr. Payne, of the firm of Mottershead and Co., Market Place, Manchester, from whom similar ones may be obtained, completely furnished, for the price of £2 2*s*. With the stand is supplied a printed card, containing directions for urine-testing.

The latter, however, are distinguished by their softer aspect and less defined outline, and they are never fibrillated at their extremities.

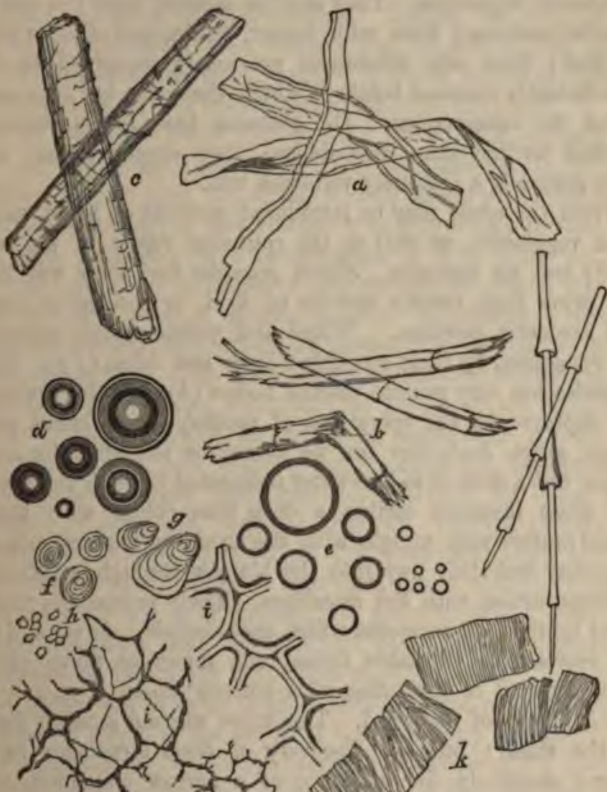

FIG. 2. Extraneous matters found in urine. *a.* Cotton fibres. *b.* Flax fibres. *c.* Hairs. *d.* Air bubbles. *e.* Oil globules. *f.* Wheat starch. *g.* Potato starch. *h.* Rice-starch granules. *i i i.* Vegetable tissue. *k.* Muscular fibres.

A few air bubbles (*d*) are generally retained beneath the covering-glass of the microscopic slide, and are apt to puzzle students. If small, they are spherical; if large, irregularly flattened. They are identified by their strong refraction, deeply-coloured thick borders and clear centres. Oil globules (*e*) are sometimes present in urine as a morbid product, in which case they are always very minute; but very often as accidental impurities. They may be derived from the use of an oiled catheter; from milk, butter, broths, and other articles of food; from oily substances previously contained in the insufficiently cleansed bottle in which the urine has been conveyed for examination. Oil globules have a less strongly marked outline than air-bubbles; they appear flatter, and have generally a distinctly yellowish tint.

From the *sputa* may be introduced portions of bread, meat, fresh vegetables, as well as the epithelial *débris* of the oral cavity and air passages. *Starch granules* find their way into the urine from certain articles of food, or the use of tooth and cosmetic powders. Wheat and potato starch granules are recognised by their concentric lines and hilus (*f g*). Rice granules are very minute cubical bodies (*h*). If the granules are ruptured by the operations of cookery (as in bread, puddings, gruel, &c.), they can no longer be identified by their forms, but a drop of iodine-water insinuated beneath the covering glass instantly strikes a deep blue colour with them. *Fæcal matters* may mingle with the urine by inadvertence, or they may find their way into the bladder through a fistulous communication with the intestines. Their presence is recognised by the food-remnants which they contain. At (*i*) and (*k*) are represented vegetable tissues and muscular fibres which were detected in the urine of a patient whom I saw with Mr. Jameson of Heywood. The urine was not sensibly fæcal to the smell; but the discovery of these structures in it proved decisively the existence of a narrow communication between the bowels and the urinary tract, and threw a strong light on an otherwise very obscure case. Particles of *soot* and *sand*, and other matters which may be designated as *dirt*, are of frequent occurrence. They are dark shapeless masses of various sizes, and all dissimilar. Any object of undefined shape, of which there are none similar to itself in the field, may almost with certainty be set down as dirt.

IV.—CHANGES IN THE URINE ON KEEPING.

The changes which take place in urine after emission are a frequent source of misapprehension. Taking the reaction of the urine as a guide, these changes may be said to take place in two opposite directions, namely, towards excessive acidity on the one hand, and toward alkalescence on the other.

1. It has been found that healthy urine, when exposed to the air, undergoes a regular series of spontaneous changes, to which Scherer[*] gave the name of *acid urinary fermentation.* The main feature of this process is a progressive increase of the acid reaction. As a consequence of this, there usually occurs, first, a precipitation of the amorphous urates, then of uric acid, and often of oxalate of lime. Frequently, likewise, confervoid vegetations, either the mould or sugar fungus, make their appearance. The acidity goes on steadily increasing for some four or five days, sometimes for a week or ten days, and then begins to decline, as the urine passes into a state of putrefaction. It now becomes opaque from the development of myriads of minute linear particles (vibrios); the odour and reaction of ammonia, together with an offensive effluvia of putrescence, become perceptible. The amorphous urate deposit will now be found changed into dark round masses of urate of ammonia; uric acid crystals give place to bright prisms of triple phosphate, and an abundant sediment of amorphous phosphate of lime sinks to the bottom of the vessel. The confervoid vegetations cease to grow with the change of reaction; and finally perish as the secretion becomes fairly putrid.

But matters do not always pass thus. Urines of low specific gravity, or of low acidity, either do not pass through this cycle of changes at all, or do so in a very imperfect manner. Their acidity undergoes no appreciable increase; and in a day or two, or even in a few hours, they become ammoniacal.

The increased acidity which occurs in the progress of the acid urinary fermentation depends chiefly on the generation of lactic acid, partly also on the production of acetic acid. Scherer believes that the mucus of the bladder acts as a ferment on the urinary pigment, and transforms it into lactic acid. Like other fermentive processes, this one is impeded or arrested by alcohol and by boiling; also by removing the ferment (the vesical mucus) by filtration.

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[*] Annalen d. Chemie und Pharm., Band 42, p. 171.

2. The changes which take place in an opposite direction, that is, towards alkalescence, are much more prone to mislead than those just described. The transformation of urea into carbonate of ammonia (*see* REACTION) is a frequent source of confusion in the examination of the urine. This transformation is brought about with great rapidity by contact with any decomposing organic matter, especially by the contact of decomposed urine. The physical and chemical characters of the secretion are then so altered, that it is unfit for clinical examination, and should invariably be rejected, except in cases where the transformation takes places within the urinary passages and a more natural specimen is therefore not procurable.

In consequence of these changes it is desirable to examine the urine within a few hours of the time of emission. Certain organic deposits are liable to be greatly altered, or altogether destroyed, by an exposure of twelve or twenty-four hours, even when the more obvious characters of the secretion have not undergone a perceptible change. Blood corpuscles, renal epithelium, renal casts, are very rapidly disintegrated, especially if the urine be of low specific gravity. On the other hand, pus, pavement epithelium, and spermatozoa resist much longer without effacement of their microscopical characters; and they may generally be recognised without difficulty in urine far advanced in putrefaction.

CHAPTER II.

PHYSICAL PROPERTIES OF THE URINE.

I.—ODOUR.

THE natural odour of healthy urine is faint and peculiar; it may be described as *urinous;* it is due to the presence of certain volatile organic acids. The addition of a mineral acid greatly intensifies, and to a certain extent modifies, the urinous odour. The sense of smell is a rough test of the presence of ammonia, and of the freshness of the secretion, or the advent of putrefaction. When urine is alkaline from fixed alkali, it has a sweetish aromatic odour like that of the fresh urine of the horse or ox. In this way the smell of the urine comes to be a ready indication of its reaction.

Certain drugs (turpentine, copaiba, cubebs), and certain articles of food (asparagus, garlic) communicate peculiar odours to the urine which lead to their immediate detection. Diabetic urine when fresh has a faint whey-like fragrance, and when fermenting it smells like sour milk. Urine containing blood or sanious discharges from the genital passages emits a stale, offensive smell, like the washings of slightly tainted flesh.

II.—COLOUR.

The colour of the urine in health is a yellowish brown. It varies in intensity from the palest straw to a full amber. The study of urinary pigments is one of great inherent difficulty; and it has been rendered truly intricate by the multiplication of new terms by successive investigators, and the confounding of pigments produced by decomposition with those really pre-existing in the urine. In drawing up the following account, I have had the advantage of perusing the manuscript of a comprehensive paper shortly to be published by Dr. Schunck of this town, who has made urinary pigments the subject of special study for several years. Dr. Schunck has placed the subject in a much clearer light than previous writers; and he has kindly permitted me to use some of his materials for the purpose of the present publication.

The colouring matters encountered in the urine may be divided into four categories, viz.:—

1. Normal pigments of healthy urine.
2. Pathological pigments due to disease.
3. Derived pigments due to decomposition of the normal pigments, or of certain colour-yielding extractives of the urine.
4. Adventitious pigments due to admixtures of bile, blood, hæmatin, pus, &c., with the urine, or to the administration of certain drugs—logwood, rhubarb, senna, santonin, &c.

1. *Normal pigments of healthy urine.*—Dr. Schunck's investigations have led him to the conclusion that the ordinary colour of normal urine is due to the presence of two substances having the properties of extractive matters. He has succeeded in separating these from one another, and from the other constituents of the urine. They have then the appearance of dark yellow syrups, being quite amorphous and deliquescent, with a peculiar, rather pleasant (not urinous) odour and a strong acid reaction which proceeds from the presence of organic acids resulting from their spontaneous decomposition. The dilute watery solutions of these extractives have exactly the same colour as urine itself.

One of these extractives is soluble in alcohol and ether as well as water. Its composition is expressed by the formula $C_{86}H_{51}NO_{52}$, and does not vary. In a long series of experiments made with urine obtained at different times and from different places, Dr. Schunck always found its composition the same. It is decomposed at a boiling temperature, yielding a large quantity of a brown resin and volatile organic acids. Its watery solution becomes several degrees darker on the addition of sulphuric or hydrochloric acid, and a brown resinous substance is gradually deposited.

The other extractive matter is soluble in water and alcohol, but not in ether. Its formula is $C_{38}H_{27}NO_{28}$. It need not necessarily pre-exist in urine ; indeed, Dr. Schunck is nearly certain that it is formed from the preceding during the process of preparation.

2. *Pathological pigments.*—The most familiar of these is a reddish-pink pigment (purpurine of Bird, and uro-erythrine of Heller) which makes its appearance in various febrile and other complaints. Purpurine has an intense affinity for uric acid and the urates, and when the latter are thrown down as a deposit it communicates to them a beautiful pink colour. Purpurine abounds in the urine of persons suffering from severe organic diseases, and especially organic diseases of the liver ; it is likewise present in all febrile and inflammatory urines. It is said to be abundant in poisoning by lead and other metals.

3. *Derived pigments.*—Schunck has shown that the normal pigments of the urine are extremely susceptible of decomposition. All strong alkaline or acid reagents, and even simple boiling, are sufficient to change them : and there is little doubt that a considerable number of the substances described by previous writers as pigments pre-existing in the urine, were, either partly or wholly, products of such decompositions. Among these may be enumerated the various *brown* and *blackish resins* of authors, the *melanic acid* of Prout, the *uræmatin* of Harley, and probably the *urochrome* of Thudichum.

The discovery of *indican* (or a substance closely resembling it) as a normal constituent of the urine by Schunck, afterwards confirmed by Carter, has thrown a strong light on the nature of some of the pigments found in urine. This substance does not itself impart colour to the secretion, but it yields by decomposition two colours well-known in the arts, viz., *indigo-blue* and *indigo-red* (uro-glaucine and urrhodin of Heller). Indigo-blue is frequently seen in putrescent urines, forming glistening blue shreds and films on the sides of the glass and the surface of the urine. Occasionally it is observed clinically. It was so noticed by Prout, who clearly indicated its nature and composition. It is also probably identical with the cyanourine of Braconnot. In the highly ammoniacal urine of cystitis I have seen on two occasions, the precipitated urate of ammonia tinted of a beautiful violet by indigo-blue. The quantity of indican in urine varies from a mere trace to a considerable proportion. Hitherto no clinical significance has been attached to its variations.

4. *Adventitious pigments.*—In jaundice the colouring matter of the *bile* is freely excreted by the kidneys, and communicates to the urine a colour varying from a saffron yellow to a dark olive-green. Bile-pigment in urine may be discovered by placing a few drops of the secretion on a white porcelain plate, with a few drops of nitric acid in juxtaposition. The two fluids are brought into contact by inclining the plate; if bile be present, a beautiful play of colours—violet, green, and red—is observed, which passes rapidly away. Bile-pigment appears in the urine before the skin is perceptibly discoloured: it also continues after the skin has attained its natural tint; so that its recognition is sometimes a useful warning of impending jaundice or a verification of a pre-existing jaundice. When a urine containing bile is kept for some days, it sometimes changes to a grass-green colour from oxidation of the biliary pigment.

Dr. Harley considers that the presence of the biliary acids in the urine is characteristic of jaundice from retention of bile, as distinguished from jaundice arising from suppression of bile. For the detection of the biliary acids he recommends that a couple of drachms of the urine be poured into a test-tube with a small fragment of loaf sugar. Then about half a drachm of strong sulphuric acid should be slowly added, in such a manner that the two fluids shall not mix. If biliary acids be present, there will be observed at the line of contact of the acid and urine—after standing a few minutes—a *deep purple hue.*[*] In a case of long-standing retention of bile from compression of the common duct by a cancerous growth of the head of the pancreas, which I recently saw with Dr. Henry Simpson, only a brandy-red colouration of the urine was produced by the application of this test.

Blood and *pus* mixed with the urine communicate to it their appropriate colours (see HÆMATURIA, HÆMATINURIA and PUS IN URINE).

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[*] Harley on Jaundice, p. 61.

Certain *medicinal* and *poisonous* substances administered internally produce peculiar alterations of colour in the urine. Creasote, and the external application of tar ointment, have been known to produce a very *dark*, almost *black* urine. In some cases of this kind which occurred in Guy's Hospital, Dr. Odling identified the dark colouring matter with indigo-blue, and he pointed out the close chemical relations between indigo and creasote.* Marcet, Prout, and Dulk have also described cases in which a black colouring matter existed in the urine. In patients taking gallic acid a dusky hue is communicated to the urine. Vogel records an instance of black discolouration of the urine after poisoning by arseniuretted hydrogen (see HÆMATINURIA.)

Rhubarb given internally colours the urine a deep gamboge-yellow, which is changed to red by the addition of ammonia; *Senna* communicates a brownish, and *logwood* a reddish tinge to the urine when administered as infusions. *Santonin* imparts a conspicuous red colour to the urine if it be alkaline.

III.—DENSITY OR SPECIFIC GRAVITY.

The specific gravity of the urine is estimated by means of the urinometer. This instrument indicates whether the urine is concentrated or dilute: and as the range of health is very great, the density does not yield direct indications of disease; nevertheless the information thus furnished is in some cases of great importance, and indicates at once the path of further research.

The usual range of density in healthy urine extends from 1015 to 1025; but it frequently mounts above or sinks below these limits. After abundant potation on an empty stomach the urine is profuse in quantity, clear, and dilute as water. Under such circumstances the density may fall as low as 1000·6, and numbers varying from 1002 to 1008 are common. Copious drinking on a full stomach has comparatively little immediate effect on the flow of urine. Prolonged fasting renders the urine concentrated.

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* Bird's Urinary Deposits, 5th ed. p. 336.

How high it is possible for the density to mount in healthy individuals it is difficult to say; but I have known it as high as 1036. With this very considerable range in health, caution must be exercised in drawing inferences from any unusual depression or elevation of the density in disease. If, however, the urine exhibit habitually, and especially in the morning before breakfast when the urine is naturally concentrated, a density below 1015, the presence of albumen in it may be suspected; if the density persist at a still lower point—1005 to 1008—the existence of insipid diabetes is to be apprehended. After hysterical paroxysms in women, after similar attacks in men, and sometimes in the apoplectic state, the urine is discharged in large quantity and of exceedingly low density.

On the other hand, a density above 1025, especially in a pale, *apparently* dilute urine, is strongly suspicious of the presence of sugar: and the higher densities, from 1035 to 1050, belong almost entirely to saccharine diabetes. Yet not exclusively so: the heaviest urine ever submitted to my examination, which had a density of 1065, did not contain a particle of sugar, but a very large quantity of albumen.

A high density in a urine free from sugar indicates concentration, and more particularly a large per-centage of urea. In the febrile state there is an absolute increase of urea, uric acid and the sulphates in the urine, with a diminished elimination of water, consequently the specific gravity ranges high. The urine has also a high density when there is rapid wasting of the tissues, especially if there be concurrent sweating or diarrhœa, in simple abstinence, after profuse perspiration from any cause, and after excessive ingestion of nitrogenised food without a corresponding use of aqueous fluids.

From the density of the urine, a rough estimate may be formed of the per-centage of solid constituents contained in it; and if the quantity voided in twenty-fours be known, the daily excretion of what may be called "solid urine" can be approximatively ascertained. Tables have been constructed on an experimental basis, exhibiting the quantity of solid matters per 1000 parts in urines of different specific gravities; and formulæ have been proposed by means of which the same result can be obtained by a simple calculation. Probably the most accurate, as well as the simplest, formula, is that proposed by Trapp. According to this, if the two last figures of the sp. gravity are doubled, the quotient represents the amount of solid matters per 1000. A thousand grains of urine, sp. gravity 1020, would therefore contain 40 grains of solids.

This method yields but rough approximations. If the urine were a solution of a single substance, or of a number of different substances in a fixed proportion to each other, the rising and falling density would indicate accurately the varying strength of the solution; but the urine is a fluid of complex composition, and its numerous constituents vary every hour in their mutual proportions, so that the results obtained in this way cannot be regarded as exact estimates. Vogel took the trouble to inquire what are the precise limits of error in this method; and he assigns them as follows: in healthy urines there is a liability to error of $\frac{1}{10}$ or even $\frac{1}{7}$; but in morbid urines, and especially those of high density, the range of error may reach $\frac{1}{5}$ and even $\frac{1}{4}$. With very multiplied observations this method certainly yields results of practical value: and it is the only one which can be used by practitioners generally. When more accurate results are required, resort must be had to the more tedious but more exact method of evaporation to dryness, and weighing the residue.

From a large number of observations by different physiologists, Dr. Parkes estimates the mean discharge of solid urine in healthy men, between twenty and forty years of age, living on good diet, at 945 grains per day.

IV.—QUANTITY OF THE URINE.

Closely connected with the specific gravity, and holding an inverse relation to it, is the quantity of the urine. The mean daily discharge ranges, in health, between 40 and 50 fluid ounces. There are, however, considerable differences between individuals. The average for some persons is only 35 ounces a day; for others as much as 67 ounces. Oscillations in the same individual on different days are also very considerable. The urine may mount to 70 or 80 ounces, or sink to 25 ounces, within the limits of health.

The flow of urine is essentially regulated by the quantity of fluids drank: controlled, however, in a most important degree by the pulmonary and cutaneous exhalation, and by the call of the system for water at the time. When the blood and tissues contain their full complement of water, any further potation results in immediate diuresis, whereby the superabundance is carried off. But when the organs are craving for more water, a large quantity may be drunk without causing diuresis. The kidneys eliminate water in strict accordance with these conditions—it being an essential and important part of their function to regulate the aqueousness of the blood.*

There is very great irregularity in the flow of urine from hour to hour as the conditions of its separation vary. After prolonged fasting the urine may sink to 2½ drachms per hour; during sleep, likewise, the urine flows slowly—at the rate of about half an ounce per hour; but after meals it rises to two or three ounces; and after drinking abundantly on an empty stomach I have seen 26½ ounces secreted in an hour; so that the stream of urine may run 85 times stronger at one time than another! It would seem, indeed, as if the kidneys (in health) supplied conditions of an almost mechanical nature, by which they were enabled to separate water at an almost unlimited rate—equal, at least, to the capacity of the gastric vessels to absorb water.

When the mode of life is equable, and the meals are taken at regular intervals, the quantity of urine secreted at different periods of the day and night follows certain tolerably regular oscillations, as is shown in the following table, which is a fair sample of a very large number of observations:—

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* The experiments on which these and the remarks which follow are based, are fully detailed in two papers by the author—one in the Memoirs of the Manch. Lit. and Phil. Soc., 1858-9; and the other in the Edinb. Med. Journ., March and April, 1860.

Breakfast at 8*; dinner at* 2. *Sleep from* 11 *p.m. to* 7 *a.m.*

Time of day.	7–8 A.M.	8–9	9–10	10–11	11–12	12–2 P.M.
	oz. dr.	oz. dr.	oz. dr.	oz. dr.	oz. dr.	oz. dr.
Hourly rate.	0 6	1 0	2 0	1 4	1 7	1 3

Time of day.	2–8 P.M.	3–4	4–5	5–6	6–7	7–9	9–11	11–7 A.M.
	oz. dr.	oz. dr.	oz. dr.	oz. dr.	oz. dr.	oz. dr.	oz. dr.	oz. dr.
Hourly rate.	1 2	10 0	2 3	2 3	2 9	1 4	1 0	0 4

A much closer insight into the varying activity of the kidneys is obtained by comparing the quantity of *solid urine* excreted at different periods of the day. The solid matters are much more constant in their quantity than the volume of the urine, which is liable to be greatly affected by potation, perspiration, &c. The annexed table contains the average results of observations made during seven days, all consecutive except one. The solid urine was calculated from the sp. gravity in the manner explained in the preceding section:—

Time of Day.	Solid Urine discharged per hour, in grains.	Diet, etc.
8– 9 A.M.	29·27	Breakfast at eight; coffee or tea, with meat and bread and butter.
9–10 ,,	39·22	
10–11 ,,	44·34	
11–12 ,,	45·24	
12– 2 P.M.	41·43	
2– 3 P.M.	38·69	Dinner at two: meat, potatoes, bread, cheese, water. (No solid food of any sort taken after dinner.)
3– 4 ,,	38·79	
4– 5 ,,	41·21	
5– 6 ,,	41·09	
6– 7 ,,	49·01	
7– 9 ,,	47·44	
9–11 ,,	37·66	
11– 1 A.M.	28·53	
1– 7 A.M.	15·53	Hours of sleep.
7– 8 A.M.	17·75	Prolonged fasting in the waking state.

The table shows in an interesting manner the increase of the renal excretion after meals, and its diminution during fasting and sleep. The increase began within the first hour after breakfast, and continued during the succeeding two or three hours; then a diminution set in, and continued until an hour or two after dinner. The effect of dinner did not appear until two or three hours after the meal; and it reached its maximum about the fourth hour. From this period the excretion steadily decreased until bed-time. During sleep it sank still lower, and reached its minimum—being not more than one-third of the quantity excreted during the hours of digestion.

All the urinary ingredients appeared to partake in the increase after meals. The urea was found more than doubled; the uric acid more than trebled; the earthy and alkaline phosphates nearly doubled.

The table shows that the vegetative functions share to some extent with the animal in the repose of sleep. The mean hourly discharge of solid urine during the waking hours, on the seven days of the table, was 33·14 grains; while the average of the hours of sleep was 15·53 grains, or less than one half. This difference is not, of course, to be wholly attributed to the effect of sleep, inasmuch as, under the arrangement of meals, during this series of observations, the period of sleep was also a time of fasting. A more exact estimate of the effect of sleep alone is obtained by comparing the urine secreted during the hours of sleep with that secreted during hours of combined waking and fasting. If we take the two last hours before sleeping (from 11 to 1), and the first hour after waking (from 7 to 8), we shall find that the mean discharge of solid urine in these three hours was 23·59 grains per hour, which is one-third more than the average of the sleeping hours.

In drawing practical conclusions concerning any deviation from the usual volume and quantity of the urine, the following points should be borne in mind.

When the urine is unusually *scanty*, it should be ascertained, before pronouncing it a morbid phenomenon, whether the patient has abstained from liquids above his habit, whether water has been eliminated in excess by some other channel, as the skin or bowels. The urine is always scanty in cirrhosis of the liver; in some forms of Bright's disease through their entire course; and in the last stage of all forms; in any condition of the heart which directly or indirectly causes passive congestion of the renal veins whereby the circulation through the kidneys is impeded. In the early stage of acute Bright's disease, the urine is very scanty, sometimes approaching or reaching total suppression. The same occurs in the collapse period of cholera. Partial or total suppression also occurs in the later stages of all organic diseases of the kidneys; and when any mechanical obstacle obstructs the flow of urine. A diminution of the urinary secretion which at all approaches suppression is of most serious consequence, and is soon followed by formidable cerebral symptoms, which bring life to a termination unless speedily relieved (see URÆMIA).

The flow of urine is *abundant* when the surface of the body is cool; also as a direct and invariable consequence of potation, unless some of the conditions already mentioned intervene.

In disease, the urine is discharged in excessive quantity in two special maladies—Diabetes insipidus, and diabetes mellitus, which will be described in a future section; also in the middle stages of atrophic degeneration of the kidneys. Temporary excess of urine occurs after hysterical paroxysms, and certain other convulsive attacks in males and females. An increased tension in the arterial system, as in some cases of hypertrophy of the left ventricle, is associated with increased secretion of urine. It is a curious circumstance, that in several organic diseases of the kidneys in which the renal substance is gradually destroyed (atrophic Bright's disease, cystic degeneration, double hydronephrosis), the volume of the urine is sometimes increased though the solid matters are diminished. This appears to be an attempt on the part of nature of a compensating character, to maintain, by excessive transudation of water, the depurating function of the kidneys under failing anatomical conditions. When at length the destruction has gone so far that this kind of compensation can no longer suffice, symptoms of fatal suppression of urine rapidly supervene.

V.—REACTION OF THE URINE.

There is no property of the urine of more varied and important significance than its reaction. Therewith is intimately connected the production of several kinds of urinary deposits, together with the origin, growth, and medical treatment of gravel and urinary calculi.

The reaction of the urine is liable to be affected by :—*food*, the *cold bath*, *medicinal substances*, *general disease*, and *decomposition* of the secretion. It is also not only important to distinguish acid from alkaline urine ; but it is at least equally so to distinguish between alkalescence from fixed alkali (potash or soda) and alkalescence from the volatile alkali (ammonia).

The most convenient method of ascertaining the reaction of the urine is by means of blue and red litmus paper. For delicate operations the violet-tinted papers are the best ; and they answer both for acid and alkaline fluids—being turned red by the former and blue by the latter. To distinguish between the volatile and fixed alkali, the paper, after being rendered blue, should be allowed to dry in the open air. If the blue colour persist after complete desiccation, the alkali is fixed ; if it disappear, and the original colour be restored, the alkalescence is due to ammonia. The smell of the urine is also a useful indication in such cases (see p. 11).

Healthy urine is generally acid. This arises chiefly from the presence of a number of acid salts—phosphates and urates ; partly also from free acids—lactic, oxalic, acetic, &c. In a number of observations by the present writer, it was found, on an average of nineteen days, that in a healthy man it required 14·10 grains of dried carbonate of soda to saturate the total daily acidity of the urine. Some days were found throughout exhibiting a feeble acidity ; on one of these only 5·9 grains of dried carbonate of soda were necessary to neutralize the whole acidity. On other days the acidity ruled high ; one day the acidity equalled 22·34 grains of carbonate of soda.

The circumstances which modify the reaction of the urine may be considered under the following headings :—

1. *Food and fasting.* Dr. Bence Jones was the first to point

out that the reaction of the urine held a close relation to the digestion of food. He found, by examining the urine at short intervals, that a notable falling off in its acidity took place after a meal; and that in numbers of healthy persons the urine became neutral or alkaline for two or three hours after breakfast and dinner. Doubts have been thrown on the conclusions of Dr. Bence Jones by Vogel, Beneke, Sellers, and Delavaud. Some years ago I undertook a series of experiments with a view of submitting this question to a fresh examination.* The urine of a healthy person was examined at hourly periods after a meal, and its acidity or alkalescence carefully determined by volumetrical analysis. My results confirmed, in the fullest manner, the observations of Dr. B. Jones. A meal, whether of animal, vegetable, or mixed food, was found invariably to depress the acidity of the urine, and in most instances to render it actually alkaline. To this movement the name of *alkaline tide* may, for the sake of brevity, be applied. After breakfast the alkaline tide was found to set in earlier than after dinner, and its duration was more brief. In forty minutes after breakfast there appeared, nearly always, a sensible declension of acidity. The urine, however, never became actually alkaline, nor even neutral, so soon. During the second hour after breakfast, the alkaline tide usually culminated; but in about a third of the observations the point of least acidity was not reached until the third hour. Then the tide turned; during the fourth hour the urine was found rapidly recovering its lost reaction, and toward the end of that time it had usually regained its previous acidity. Thus for about four hours breakfast exercised a depressing effect on the acidity of the urine; but the secretion was not actually alkaline usually for more than an hour, sometimes for two, and very rarely for three hours.

The effect of dinner was not perceptible until the second hour after the meal. During the succeeding three hours (third, fourth, and fifth hours) the alkaline tide ran in its greatest strength. In the third and fourth hours the urine was always (with two exceptions) alkaline, when the meal had been of mixed food or animal diet. At the end of the sixth hour the tide had generally turned, and the acid reaction been restored. Three hours was the usual duration of the alkalescent state after dinner; sometimes two hours; more rarely four hours; and on one occasion five hours. The amount of free alkali discharged after dinner was generally nearly double the quantity after breakfast. This was due, probably, simply to the fact that breakfast was a much lighter meal than dinner, and its impression on the system consequently less.

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* See a paper by the author, entitled, "A Contribution to Urology," in the Memoirs of the Manchester Lit. and Phil. Soc., 1859.

The alkaline urine passed after food owed its reaction to fixed alkali, and not to ammonia. It was rich in alkaline and earthy phosphates; on these latter, in a basic state, depended apparently its alkaline reaction. Sometimes it was clear when voided, but more commonly turbid, from the precipitation of earthy phosphates.

Although the immediate effect of a meal was thus to depress the acidity of the urine, the more *remote consequence* was to maintain and even increase the acidity. This was seen most distinctly when comparison was made of the acidity of the morning urine when supper had been taken the night before, with that of the morning urine when no supper had been taken. In the former case the free acid discharged in the hour preceding breakfast was enough to saturate 0·88 grains of dried carbonate of soda: whereas on the mornings after supperless nights the discharge of acid was only equal to 0·51 grains.

The remote effect of animal food appeared considerably greater than that of vegetable food: so that a highly animalized diet would tend in the long run to intensify the acidity of the urine—a conclusion quite in harmony with ancient opinion.

Clinically, the urine is rarely observed to be alkaline after food. For, although it may be alkaline as it leaves the kidneys during several hours a day, after the two principal meals, it is mixed in the bladder with acid urine secreted before and after the alkaline tide, and the whole product ejected by micturition is acid. It is necessary therefore, in order to test the effect of a meal, to analyse the secretion, as it were, by examining it at hourly intervals. It happens occasionally, however that the urine of an ordinary micturition is the isolated product of the alkaline tide. I have known even a calculous patient, whose urine habitually deposited large quantities of uric acid, to void an alkaline urine in the forenoon from the effect of breakfast.

Dr. B. Jones considered the depression of the acidity of the urine after a meal, to depend on the withdrawal of acid from the blood into the stomach for the purposes of digestion; whereby the blood became for the time less capable of yielding acid to the kidneys. On the completion of digestion the gastric juice was re-absorbed with the chyle and presently communicated its acid to the urine. An antagonism was thus supposed to exist between the stomach and kidneys: when the stomach was empty its mucous membrane was neutral, while the urine on the contrary was highly acid; but when the stomach was full, acid gastric juice was abundantly poured out on its mucous surface for the purposes of digestion, and at the same time the urine tended towards neutrality or alkalescence.

While admitting the probability of some such correspondence, I am disposed to attribute the occurrence of the alkaline tide after meals to a totally different cause—namely, to the entrance of the newly digested food into the blood. If, as is believed, the normal alkalescence of the blood is due to the preponderance of alkaline bases in all our ordinary articles of food, a meal is *pro tanto* a dose of alkali, and must necessarily, for a time, add to the alkalescence of the blood; and as the kidneys have delegated to them the function of regulating the reaction of the blood, the urine immediately reflects any undue addition to, or subtraction from, the blood's proper alkalescence. This hypothesis is mainly supported by the coincidence of time which exists between the passage of the digested food into the blood and the occurrence of the alkaline tide. The gastric juice is poured into the stomach *immediately* after a meal, but the acidity of the urine does not suffer depression for an hour or two afterwards—not in fact until the meal has been in great part absorbed.

After the primary effect of a meal has passed off, the acidity of the urine slowly increases until food is taken again. The highest acidity is, therefore, always found after the longest fasting, or just before meals. In the early morning before breakfast, the urine was always found excessively acid, and deposited abundance of urates on cooling. There seems, however, a limit to the increase of the acidity after prolonged fasting: Dr. Bence Jones found that continuing to fast for twelve hours beyond the usual time of taking food did not intensify the acidity of the urine.

2. *Effects of Medicines.* Both mineral and vegetable *acids* when administered in large quantities tend to raise the acidity of the urine; but their effect is inconsiderable. Urine that is habitually alkaline can certainly not be rendered acid by the internal administration of acids even in very large quantities.* The most powerful acidifiers of the urine are probably free carbonic acid (Heller) and benzoic acid; the latter appears in the urine as hippuric acid.

Alkaline substances have a much more powerful influence; and it is an easy matter to deprive the urine of its acid reaction and to render it strongly alkaline at pleasure. This effect may be attained by the caustic and carbonated alkalies, or by the alkaline salts of a certain group of vegetable acids—acetic, tartaric, citric, malic, and lactic acids. The most convenient for the purpose, as well as the least disturbing to the digestive organs, are the bicarbonates of potash and soda, and the acetates and citrates of the same bases. By the administration of these salts the urine may be kept persistently alkaline for weeks and months without detriment to the general health. It requires from 300 to 400 grains of the bicarbonate of potash, and about as much of the acetate and citrate, given in divided doses during the twenty-four hours, to keep the urine steadily alkaline in the adult. From numerous observations on different individuals, I found, that, given in these large doses, about two-thirds of the alkali appeared in the urine as free carbonate; while the remaining third was expended in neutralising the acidity of the urine and otherwise disposed of. The conversion of the acetates, citrates, etc., into carbonates, which was shown long since to occur by Wöhler, takes place, according to Buchheim and Magawley, on the intestinal canal, and the salts in question are therefore absorbed into the blood as carbonates. The bi-carbonates, acetates, and citrates, if moderately diluted, were not found to have any tendency to cause diarrhœa : the tartrates, on the other hand, were always found to occasion more or less purging.

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* For further information on the action of acids on the urine, see Parkes, "On the Composition of the Urine," p. 145 *et seq.*

The basic phosphate of soda, the common phosphate of soda, and borax, likewise possess the power of alkalising the urine ; but their effect is very feeble compared with that of the salts before-mentioned. The common phosphate of soda, in the quantity of 640 grains, in the twenty-four hours, in divided doses, produced a total alkalescence of the urine, only equal to 22 grains of carbonate of soda: whereas half the quantity of the acetate of potash produced an alkalescence equal to 120 grains of carbonate of soda: 640 grains of the basic phosphate of soda produced an alkalescence equal to 37 grains of carbonate of soda : 320 grains of borax gave an alkalescence of 9 grains of carbonate of soda ; this last salt proved difficult of toleration by the stomach.

The power of alkalising the urine is especially valuable in the treatment of urinary gravel and calculi; and to the chapter on the solvent treatment of urinary concretions I must refer the reader for further details on the subject.

3. *The Cold Bath.* Duriau* found that the urine became invariably alkaline after prolonged immersion of the body in a bath at a colder temperature than that of the body. Even the addition of nitric acid to the bath did not in the least alter the result ; nor did the addition of carbonate of potash cause an increased alkalescence.

4. *General Disease.* Frequent or persistent alkalescence of the urine, from fixed alkali, is an uncommon condition in any class of complaints : but a series of such cases have been recorded by Dr. Bence Jones,† and I have observed a considerable number myself. In persons of debilitated constitutions, in the anæmic state which sometimes follows subacute rheumatism and gout, in chlorosis, atonic dyspepsia, chronic vomiting, and even in chronic phthisis, I have seen the urine present this

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* Archives Générales. 1856. I. 167.

† Med. Chir. Trans. Vol. xxxv.

character. Generally, the alkalescence came and went capriciously; continuing for two or three days, and then disappearing; but presently returning again. Sometimes, however, the urine remained steadily alkaline for many weeks without intermission. In one case of this kind—a phthisical patient—the urine became acid on the occurrence of an attack of erysipelas of the head and face: it remained acid during the attack, and after its subsidence became again alkaline.

The clinical significance of alkaline urine from fixed alkali is by no means serious; it is not associated with any special morbid state, but is an occasional accompaniment of debility and spanæmia, from whatever cause arising. It is to be remembered, however, that there is a rare variety of urinary calculus composed of phosphate of lime, which must be caused by some such condition of urine as this. Individuals passing an alkaline urine are generally suitable subjects for a tonic and stimulating treatment: and, if otherwise permissible, exercise in the open air.

5. *Ammoniacal Urine. Decomposition of Urea.* The importance of distinguishing between urine which is alkaline from fixed alkali, and that which is alkaline from ammonia, has already been insisted on. The two conditions are contrasted, not only chemically, but equally so pathologically and clinically.

Urine which is alkaline from *fixed* alkali is always secreted alkaline by the kidneys; it deposits, if at all, simple amorphous phosphate of lime, of which the particles have no tendency to accrete into gravel or calculi; it has a sweet aromatic odour; it is perfectly bland and innocuous to the mucous membranes, and is not associated with inflammation of the urinary passages.

Ammoniacal urine, on the other hand, is only in the rarest instances and in the gravest circumstances, secreted ammoniacal by the kidneys, but usually becomes so by an after change in the lower urinary passages; it deposits a mixture of the amorphous phosphate of lime and crystals of the ammoniaco-magnesian phosphate: this deposit has a strong tendency to aggregate into masses or concretions; the urine has an ammoniacal and often an offensive putrescent odour; it is highly irritant to the mucous membranes, and excites inflammation of them if the contact be long continued.

A urine alkaline from fixed alkali reflects a state of the blood; an ammoniacal urine points to a local affection of some part of the lower urinary passages. This latter statement is, however, not absolutely without exception. In two instances (both cases of advanced Bright's disease) I have observed the urine to be ammoniacal as it flowed from the bladder without any clinical or *post-mortem* sign of inflammation of any part of the urinary passages, or any evidence of such delay in the evacuation of the urine as might determine decomposition of it in the bladder. One of these cases is referred to hereafter (see BRIGHT'S DISEASE). Dr. Graves (Clin: Lects. 1, p. 272) gives also two cases, one of continued fever, and the other of anasarca and ascites, in which the fresh urine contained large quantities of carbonate of ammonia without the least evidence of decomposition after secretion.

The mode in which the urine becomes ammoniacal from decomposition is easily explained. One atom of urea with two atoms of water, by a simple re-arrangement of their particles, become converted into two atoms of carbonate of ammonia: 1 at. urea $C_2H_4N_2O_2 + 2\ HO = 2\ (NH_3CO_2)$. This change is so easily brought about that mere boiling of a solution of urea in distilled water is sufficient to effect it. Urea in urine is decomposed at the ordinary temperature of the air by the action of any putrescent substance, and by none more quickly than decomposing urine. I have already remarked on the facility with which urine may become ammoniacal after emission when it contains any organic matter —pus, blood, epithelium, albumen, etc.: also when it is very dilute and feebly acid or alkaline.

If the urine be ammoniacal *when voided*, this is nearly always associated with inflammation of some part of the urinary mucous membrane—generally that of the bladder. Any condition which interferes with the complete emptying of the bladder in micturition favours the production of ammoniacal urine. Consequently, injuries to the spine determining paraplegia with paralysis of the bladder, obstinate urethral stricture, enlarged prostate, calculous concretions, morbid growths or foreign bodies in the bladder, are sooner or later complicated with ammoniacal urine. A very distressing and intractable state of things is thus brought about. The ammoniacal state of the urine irritates the mucous membrane and induces cystitis: and the purulent secretion thus engendered reacts on the urine and favours its decomposition. The two conditions mutually aggravate each other and perpetuate each other's existence after the original cause has passed away. Cystitis may, in this way, persist for years after the removal of a stone, or the cure of a stricture, which was its original cause.

Dr. Owen Rees believes that the urine is sometimes alkaline from the secretion of an alkaline mucus by the mucous membrane of the bladder. When the membrane is irritated or inflamed, as in paraplegia from spinal injuries, extroversion of the bladder, etc., the irritated membrane pours out, as he believes, so much alkaline mucus that the reaction of the urine is changed thereby. Dr. Rees fortifies this hypothesis by an observation which he made on a case of extroverted bladder. He says: "As is usually the case in such persons the anterior portion of the bladder was wanting; so that the fundus of that viscus covered by mucous membrane was projected forward where the abdominal walls were deficient. The openings of the ureters were thus presented to view. The mucous membrane was red and inflamed from exposure, and an alkaline fluid was constantly discharging from its surface. To what this alkaline flux amounted during the day it was of course impossible to ascertain; but it was more than sufficient to destroy the acidity of the urine, which was quite alkaline after flowing over the membrane. Thus a piece of blue litmus was applied to the openings of the ureters, so as to test the urine immediately it flowed from them: the paper was reddened, indicating that the urine was secreted of its natural character, and with its full amount of acidity. When, however, the litmus paper was applied about a quarter of an inch below the opening, so as to test the urine after it had passed over that short distance of mucous surface, its character was quite changed; it no longer reddened the blue litmus paper, but on the contrary was sufficiently alkaline to restore the blue colour to those parts of the paper which had been previously reddened by exposure to the urine as it escaped fresh from the ureters."*

An opportunity occurred to me of repeating this observation on a patient with extroversion of the bladder; but I was not able to satisfy myself that the alkalinity of the exposed mucous membrane was not owing to blood-serum or lymph which oozed from the raw excoriated surface, rather than to any mucous secretion such as might be yielded by a merely inflamed mucous membrane.

The therapeutical indications in cases of ammoniacal urine from decomposition within the bladder, are clear enough. The first object is to remove, if possible, the impediment to the complete emptying of the viscus. In the case of a stone or foreign body in the bladder, and in stricture, this is within reach of surgical operation. If the cause be irremovable, or if the ammoniacal urine and cystitis persist after the removal of the original cause, all our efforts must be directed to prevent the sojourn in the bladder of the stale remnants of urine after micturition; and this can only be thoroughly effected by washing out the bladder, by means of a double catheter, with a pint or two of warm water once or twice a-day. There is however another mode of washing out the bladder which I have several times resorted to with advantage in a certain class of cases, more particularly in those in which chronic cystitis is kept up by the ammoniacal state of the urine, after the original cause has been removed. In these cases the patients are made to drink large quantities of diluents at regulated intervals. An abundant flow of very dilute urine is thereby kept up which effectually washes out the bladder and gradually restores the urine to its natural state.

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* Lettsomian Lectures, Med. Times and Gaz. 1851.

CHAPTER III.

CHEMICAL CONSTITUENTS OF THE URINE AND THEIR VARIATIONS—INORGANIC DEPOSITS.

I.—PRELIMINARY REMARKS ON URINARY DEPOSITS AND THEIR CLASSIFICATION.

A VERY scanty, light, cloudy deposit is natural even to the healthy urine after standing some hours. This usually sinks to the bottom; but occasionally it floats like a cloud about the middle or near the surface. It is composed of epithelial scales (or remnants of them) from the mucous surfaces of the bladder and urethra, mingled with pigmentary particles of uncertain source (see chap. iv.). Of mucus, having the usual glairy character, there is no visible trace in perfectly healthy urine.

Under a variety of unnatural circumstances more abundant deposits or sediments occur in urine ; and a knowledge of their nature sometimes yields most important practical information.

Urinary deposits are arranged in two divisions—*Inorganic* and *Organic*.

Inorganic deposits include substances which, for the most part, exist naturally in the urine in the soluble state; but which, owing to their excessive quantity, or a change of reaction in the urine, or some other circumstance, are rendered insoluble, and thereby precipitated in a crystalline or amorphous condition. This division contains :—Uric acid, the amorphous urates, urates of ammonia and soda, oxalate of lime, ammoniacal and earthy phosphates, carbonate of lime, cystine, leucine, and tyrosine. All these are *soluble* in mineral acids or in alkalies; and one of them (the amorphous urate) by simply warming the urine.

Organic deposits embrace all those organic forms, of which the presence alone in urine is sufficient, from their insolubility, to determine their subsidence. They do not belong in any proportion to the healthy secretion; and whenever introduced, they are merely suspended in it; so that when the urine is left at rest, they gravitate to the bottom and form a sediment. This group includes in addition to the pigmentary particles already alluded to, epithelial cells from the uriniferous tubes or from any part of the genito-urinary passages, casts or moulds (composed of a fibrinous matter) of the uriniferous tubes, oily particles, pus, blood, cancerous and tuberculous *débris*, spermatozoa, and confervoid growths. All these are *insoluble* in acids and alkalies as applied in the ordinary examination of the urine.

II.—URIC ACID. $C_{10}H_4N_4O_6 + 4\ Aq.$

(*Synonym—Lithic acid.*)

Uric acid exists in normal urine in combination with alkaline bases; but under certain conditions it is precipitated in the free state, and forms a deposit of orange-red crystals.

Naked-eye characters.—The crystalline nature of the deposit can nearly always be recognised by the naked eye; but in rare instances the crystals are so small that they require the microscope for their detection. Uric acid crystals may form a film on the top, or lie scattered as brilliant brown specks on the sides of the glass, or subside into a dense red deposit like cayenne pepper. The naked eye is nearly always sufficient to identify uric acid with certainty, because no other *brown crystals* occur in urine as a spontaneous deposit. When the crystals are very minute, the deposit resembles the amorphous urate, but is denser, and sinks more rapidly. Urine depositing uric acid has commonly a rich yellow or orange colour, and is invariably acid.

Micro-chemical characters.—The primary form of uric acid is a rhombic prism or lozenge, and to some modification of this figure the protean diversities of uric acid crystals may all be referred. The angles of the crystals are sometimes almost equal, and then quadrangular tables or almost perfect cubes are obtained (Fig. 3, *a b*).

More frequently the angles are rounded off (*c d*) so as to produce ovoids and barrel shapes. A still greater elongation produces a rod, and when a number of these are joined together in a common centre, stars are produced. The beauty and endless variety of these stars are marvellous, and render them seductive microscopic objects (Figs. 4 and 5).

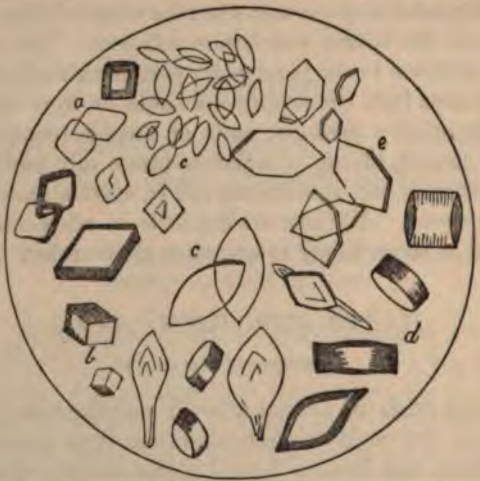

FIG. 3. The simpler forms of uric acid crystals — quadrangular and oval tablets, cubes, six-sided tables, lozenge and barrel-shaped figures.

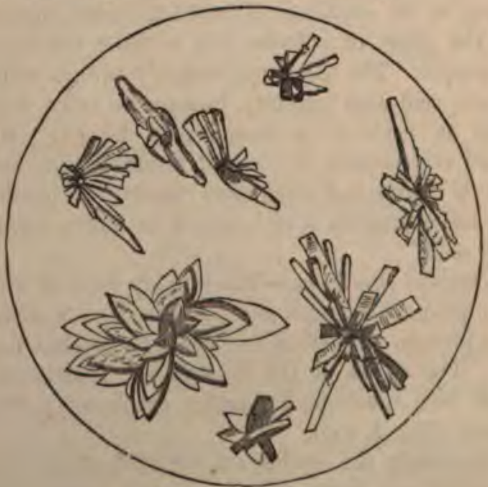

FIG. 4. Stars of uric acid.

Sometimes the rays extend only in one direction and a fan-shaped figure is produced, or two fans are joined in a common centre (Fig. 5).

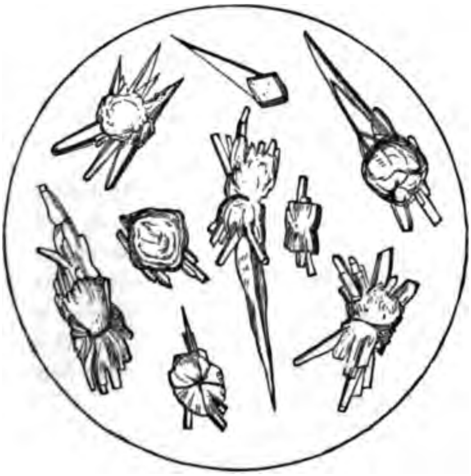

FIG. 5. Stars and spikes of uric acid.

Among the less common varieties may be mentioned pointed, solid-looking crystals with a dark shading at either end (Fig. 6, *a*).

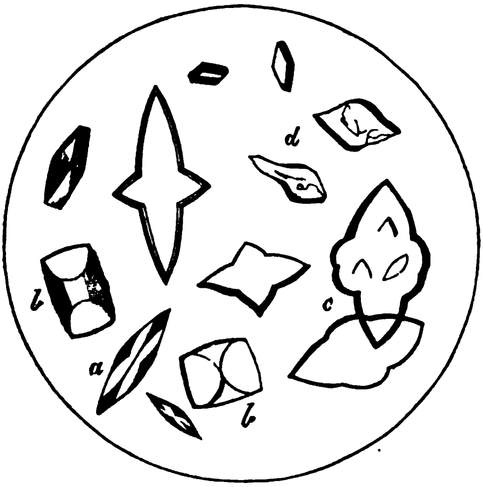

FIG. 6. Rarer forms of uric acid crystals.

When these lie flat they have a totally different appearance, and resemble prisms of the triple phosphate (*b b*). Other forms are halbert-shaped (*c*), six-sided tablets (Fig. 3, *e*), &c. The most curious and varied forms of uric acid are generally found in albuminous urines.

Uric acid is excessively insoluble. It requires 1800 parts of boiling water and 15,000 parts of cold water for solution. It is insoluble in all dilute acids, but is decomposed with effervescence by strong nitric acid. Caustic alkalies dissolve it readily, especially with the aid of heat. It dissolves also freely in weak solutions of the carbonates of potash and soda, and in solutions of borax and common phosphate of soda. It is insoluble in alcohol and ether. It is entirely dissipated by a red heat. The most delicate mode of recognising uric acid is by the murexid test. This is performed by taking a small quantity of the suspected substance and placing it on a porcelain dish or a slip of glass : a couple of drops of strong nitric acid are then added and the heat of the spirit-lamp applied; the uric acid dissolves with effervescence; the heat is continued until the liquid dries into a yellowish-red residue. If the residue when cool is touched with a rod dipped in caustic ammonia, a bright violet hue (murexid) is instantly developed, which is perfectly characteristic.

Origin and occurrence.—The quantity of uric acid in the urine is very minute; and were it not for its sparing solubility and liability to be precipitated both before and after emission, its clinical significance would be very small. The daily excretion of uric acid amounts to no more than 8 or 10 grains. Individuals vary a good deal in the amounts which they habitually separate. In three healthy young students living on a similar diet and under similar circumstances, I found the following numbers :—

No. 1 (mean of 47 days)	8·051 grains.
,, 2 (mean of 5 days)	3·462 ,,
,, 3 (mean of 3 days)	6·171 ,,

Dr. Hammond found in his own case the daily average as high as 14·14 grains.

The excretion of uric acid also presents considerable variations in the same individual from day to day. The greatest oscillation of this sort observed by myself, amounted to a difference of more than one-half on two successive days: on the first day 5·45 grains were separated, and on the following day 11·7 grains. It was found that when the mode of life was tolerably uniform, the amounts separated in periods of five consecutive days varied only slightly from each other in the same individual.

The occurrence of a spontaneous deposit of uric acid is by no means a sure indication of an increased excretion; and I frequently found that those days on which a spontaneous deposit occurred, showed less uric acid than those days on which no uric acid was spontaneously deposited. The mean daily quantity of uric acid separated in twelve days on which there was a deposit, was 7·7 grains; and the mean of twenty-five other days on which no uric acid was spontaneously deposited was 7·3 grains.

The digestion of food has a marked effect on the excretion of uric acid. I found it increased after eating, not only absolutely, but also relatively to the other solid matters of the urine. In the following table the results of seven days' observations on the effect of dinner are exhibited. Three periods are chosen for comparison, namely:—1, during the prevalence of the alkaline tide which corresponds with the entrance of the digested food into the blood; 2, during the subsequent period in which the acidity of the urine is restored, but the effect of the meal still continues to be perceptible in the considerable quantity of solid matters separated by the kidneys;* and 3, during sleep, which is also a time of fasting.

Time of day. (Dinner at 2 P.M.)	Uric acid, per 1000 grains of liquid urine.	Uric acid per hour.	Uric acid per 100 grains of solid urine.
4– 7 P.M., alkaline tide.	0·40 grains.	0·36 grains.	0·83 grains.
9–11 ,, acidity restored.	0·18 ,,	0·13 ,,	0·34 ,,
1– 7 A.M., urine of sleep.	0·39 ,,	0·10 ,,	0·60 ,,

It is seen that the absolute quantity hourly secreted is three times greater during the period of the alkaline tide than during the other periods; its proportion to the total solids is also very sensibly greater. Even its proportion to the water of the urine is greater than at any other period, though the urine of sleep generally (under the mode of life then followed) deposited amorphous urates very copiously after standing a few hours, whereas the urine of the alkaline tide never deposited urates.* It is further seen from the table that the amount of uric acid has no relation to the degree of acidity of the urine.

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* See Table, p. 19.

Professor Ranke has shown that neither sex nor age, nor the height and weight of the body, have any decided relation to the daily excretion of uric acid.† The season of the year, and the animal or vegetable nature of the food, have little influence, provided the articles of diet are equally rich in nitrogen. The effect of exercise is uncertain; sometimes it increases, sometimes it diminishes the uric acid.‡

Pathologically, it is found that the daily excretion of uric acid is markedly increased in the febrile state, in certain diseases of the liver, and after an attack of gout. On the other hand, it diminishes during the paroxysm of gout, and according to Ranke, after large doses of quinine.

Uric acid is nearly related, both chemically and physiologically, to urea. Uric acid yields urea as one of the products of its decomposition, both by artificial means in the laboratory and within the animal body. Nevertheless, the most exact observations have failed to show that there is any inverse correspondence between the excretion of the two substances; usually urea and uric acid increase and diminish together.

Clinical significance of uric acid.—From what has been already stated, it will be readily conceived that the clinical interest of uric acid has not so much to do with the variations of its quantity, whether absolute or relative, as with its precipitation in the free state, and the time and place of that precipitation. The circumstances favourable to the precipitation of free uric acid are, an acid reaction of the urine, and abeyance of the conditions which determine the precipitation of uric acid in combination (amorphous urates); these latter are considered in the next section.

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* The seven days' experiments here spoken of are the same seven days which are tabulated at p. 19.

† Ranke—Ausscheidung d. Harnsäure beim Menschen, Munich, 1858.

‡ See Parkes On the Composition of the Urine, p. 88.

A deposit of uric acid occurring some twelve or twenty hours after emission has no pathological signification. Healthy urines usually deposit uric acid as a normal event in the course of the acid urinary fermentation (see p. 9). If the deposit takes place within three or four hours after emission, the circumstance is certainly not natural; but it is not one requiring special therapeutical attention: it is frequently observed in convalescence from febrile complaints, especially articular rheumatism; also in the middle periods of chronic Bright's disease, in chorea, and in certain types of diabetes.

But if uric acid be precipitated before the urine cools, or immediately after, it cannot fail to awaken apprehensions that a similar event may take place within some part of the urinary passages, and give rise to the formation of gravel and calculi, with all their train of painful and dangerous consequences. A prophylactic treatment is urgently called for under such circumstances, by which this danger may be warded off. But it will be more convenient to postpone the further consideration of this important subject to the sections which are specially devoted to the pathology and treatment of calculous disorders.

III.—AMORPHOUS URATES.

(Synonyms—amorphous lithates; urate of ammonia of Prout and Bird; urate of soda of Heintz and Lehmann; lateritious deposit.)

Naked-eye characters.—The "amorphous urate" usually occurs as a loose, reddish, pulverulent deposit wholly devoid of any approach to crystallization. Its colour is always deeper than the urine from which it falls; but the colour varies extremely both in intensity and tint. It may be fawn, orange, brick-red, pink, or purplish. It commonly sinks soon and completely; more rarely, especially in albuminous urines, the precipitate continues a long while diffused in the urine, giving it a milky appearance. If the precipitation takes place after the urine has been at rest in the urine-glass, a film or bloom forms on the surface and sides, which is readily seen by inclining the glass to one side. By this peculiarity the amorphous urates may be distinguished from all other urinary deposits by the unaided senses.

Micro-chemical characters.—Under the microscope the deposit is found to be composed of minute particles or granules, coarser or finer, and more or less opaque, according to the closeness of its aggregation (see Fig. 7).

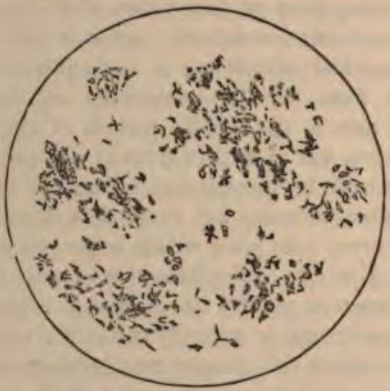

Fig. 7. Amorphous urate deposit.

By warming the urine, the amorphous urate dissolves; the light-coloured and looser deposits disappear with a slight heat, but the deeper coloured and denser ones require a more elevated temperature. As no other urinary deposit disappears with simple heat, this circumstance offers an easy means of recognition. The amorphous urate answers to the murexid test for uric acid. It is decomposed by the vegetable and mineral acids (though only slowly in the cold by the former), and uric acid crystals are deposited, which may be recognised under the microscope. The urates dissolve in the caustic alkalies, and in solutions of the carbonates of potash and soda. They possess an intense affinity for the brown and pink pigments of the urine, which they carry down with them when precipitated; and the varied tints which they present as deposits depend on this circumstance.

The chemical composition of this deposit has been a subject of much dispute. Prout and Bird believed it to be composed of urate of ammonia, and it usually passes under that name in this country. In Germany it is commonly considered to be mainly composed of urate of soda. More recent observations indicate that neither of these views is correct; it would appear rather that the amorphous urates have not a fixed and constant composition, but vary considerably in different samples. In all, however, uric acid is combined with several bases—potash, soda, ammonia, and lime; and this is the special chemical characteristic of the deposit, that it is composed of *mixed urates*. Sometimes one base and sometimes another preponderates. The proportion of uric acid in the deposit is very large, but not constant. Scherer found a little over 80 per cent.; Dr. Bence Jones over 90 per cent. This proportion is about twice as much as is necessary to form acid urates (bi-urates) with the bases present; so that about one-half the uric acid is loosely united with the bi-urates to form the deposit, which therefore resembles in its chemical constitution the quadroxalate of potash. The loosely combined uric acid can be separated from the associated bi-urates by simply treating the deposit with warm water, or by repeatedly washing it on a filter with cold water.

Dr. B. Jones found potash the most abundant base, next ammonia, and last soda, as the following table of his analyses shows:*—

	1st Analysis.	2nd Analysis.
Uric Acid	94·36	91·06
Potassium	3·15	3·78
Ammonium	1·36	3·36
Sodium	1·11	1·87

Hassall and Scherer always found lime in not inconsiderable quantity. Dr. Bence Jones succeeded in producing artificially exact counterparts of the amorphous urates both with potash and soda.

The precipitation of the amorphous urates depends on a conjunction of the following conditions: an acid reaction, low temperature, and concentration of the urine. The occurrence of this deposit is a sure sign of an acid reaction; and the more acid the urine, the more liable is it to deposit the amorphous urates. A drop of acetic or nitric acid will frequently determine at once the precipitation of the amorphous urates in a previously clear urine; and when urine becomes sedimentary after twelve or twenty-four hours, this is due to the increased acidity produced by the acid urinary fermentation. The effect of temperature is very marked: and on cold winter mornings the urine voided on getting out of bed generally becomes turbid from precipitated urates a few hours after.

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* See a Paper by Dr. Bence Jones in the Journal of the Chemical Society, June, 1862.

The amorphous urate deposit is not a sign of excessive secretion of uric acid by the kidneys; it indicates rather, that its proportion to the water of the urine is excessive. Urines of a high density, provided their reaction be acid and the temperature low, usually deposit urates in healthy persons. There is this difference between the conditions favourable to the deposit of free uric acid and of the amorphous urates—that a high density (or concentration) favours the latter, and a low density (or dilution) favours the former. On a comparison of the densities of a large number of urines, depositing respectively amorphous urates and free uric acid, I found the mean sp. gravity of the former 1027 and of the latter 1021. It is familiarly observed that a urine which throws down lithates, will begin to deposit free uric acid a few hours after, when it has become, *quoad* uric acid, less concentrated.

The clinical significance of a urate deposit can be appreciated only after due consideration of the above physical and chemical conditions of its occurrence in the physiological state. I have already stated that no conclusion as to excessive elimination of uric acid can be drawn from the occurrence of the urate deposit. It has also been shown in a previous page, that during the absorption of food, and the flow of the alkaline tide, the excretion of uric acid is at its maximum, though the urine at this period very rarely deposits urates, owing to the depression or disappearance of its acidity; and conversely, that after long fasting the urine is very apt to deposit urates, because it is then concentrated and highly acid, though the hourly rate of excretion of uric acid is then at its lowest ebb.

A deposit of amorphous urates may be regarded as having either a physiological or a pathological signification. *Physiologically*, a urate deposit may be expected after profuse sweating, violent exercise, prolonged abstinence from food and drink, and in cold weather. Under these circumstances the deposit is occasional, and its colour usually pale fawn. *Pathologically*, the most common determining cause of the precipitation of the amorphous urates is the febrile state. Even a slight degree of pyrexia, as in a common cold, is usually accompanied with a urate deposit.

The frequent or constant occurrence of a lithate deposit without, or with only a feeble degree of pyrexia, is a circumstance to awaken suspicions of some serious organic disease; but the indication is more general than special. Organic disease of the lungs, heart, liver, spleen, or any other part, attended with emaciation and waste of the tissues, is usually accompanied with abundant urate deposit.

Functional derangements of the digestive organs are also generally accompanied by pale lithate deposits in the urine. Their occurrence depends, in many cases at least, as Dr. B. Jones has indicated, on a connection between the reaction of the mucous membrane of the stomach and that of the urine (see p. 25).

Treatment.—From what has been stated of the determining conditions of the amorphous urate deposit, it is evident that it seldom requires direct treatment. Its indications are of more service in diagnosis and prognosis than in therapeutics. Sometimes the persistence of a lithate deposit occasions such alarm to the patient that it may serve a good purpose to cause it to disappear, though no really curative end may be gained thereby.* This is easily and harmlessly effected by a few two-scruple doses of citrate of potash. When this direct purpose is not aimed at, the treatment must be directed to the removal of the condition causing the deposit.

IV.—CRYSTALLINE URATES.

Urate of soda and urate of ammonia are sometimes deposited separately in urine, in the crystalline form, and under circumstances wholly different from those which determine the precipitation of the amorphous urates.

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* I have known a physician summoned from London to Manchester, at a fee of 120 guineas, simply on account of the alarm of a feverish patient and his friends concerning a simple amorphous urate deposit.

Urate of Soda.—Urate of soda is familiarly known as a constituent of gouty concretions. When the point of a lancet is thrust into one of the yellowish-white nodules so common on the ears of gouty persons, a whitish mortar-like matter escapes, which, under the microscope, is resolved into myriads of long delicate needles, arranged in bundles or stars, or lying separately (Fig. 8, *a a*).

These acicular forms are never deposited spontaneously in the urine: but they may be readily produced by adding a little liquor sodæ to the common amorphous urate, in a watch-glass, and allowing the solution so formed to concentrate by evaporation in the air (Fig. 8, *b b*).

Urate of soda is a comparatively rare spontaneous deposit in urine. It occurs, however, occasionally in gout, and in the febrile state, especially in children. It forms a whitish or yellow sediment, which sinks rapidly; it is associated with an acid reaction of the urine, and is frequently, if not generally, deposited in the bladder before the emission of the urine. In this respect it differs from the amorphous urate, which is never deposited until the urine has cooled.

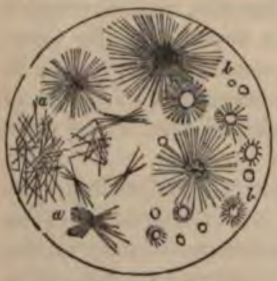

FIG. 8. Urate of soda.
a a. From a gouty concretion; *b b*. Artificially prepared by adding liq. sodæ to the amorphous urate deposit.

Under the microscope it exhibits irregular, opaque, globular and lumpy masses, from which project spiny crystals, sometimes straight, sometimes variously curved (see Fig. 9).

The occurrence of this deposit in the febrile complaints of infants and children probably depends on the urine being excessively scanty and concentrated and long detained in the bladder. Its appearance in such cases is temporary, and ceases on the re-establishment of the flow of urine. The annexed drawing (Fig. 9) was made from a deposit voided by a little child of three years. The child was suffering from severe infantile remittent, and no urine had been passed for two days. While I was examining the abdomen, the child cried, and the urine began to flow. The first portions were turbid and of a gamboge yellow colour, and contained the spiny masses here delineated; after about an ounce of this had come away, several ounces of clear high-coloured acid urine followed.

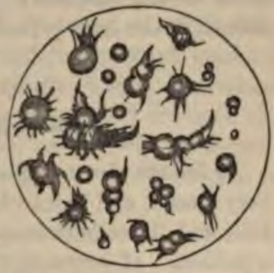

Fig. 9. Hedge-hog crystals of urate of soda, spontaneously deposited from the urine of a child.

Clinically, this deposit derives its chief importance from the circumstance that it is precipitated within the urinary passages. The spiny crystals irritate the mucous membrane of the bladder or urethra; and the latter canal may even be blocked up by impaction of masses of the deposit. It may also form a nucleus around which calculous matter may hereafter aggregate. The great comparative frequency of vesical calculi in children is not improbably owing to the occurrence of this deposit in the numerous fugitive febrile attacks to which children are subject.

Urate of ammonia.—When urine becomes strongly ammoniacal, it is liable to precipitate urate of ammonia, in addition to the mixed phosphates which are necessarily deposited under those circumstances. The urate of ammonia has usually a dense white colour; but I have known it possess a beautiful violet hue.

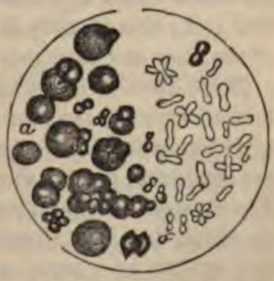

Fig. 10. Urate of ammonia spontaneously deposited. *a.* Spheres and globular masses; *b.* Dumb-bells, crosses, rosettes.

Two forms are seen under the microscope. The most common are spheres and globular masses, which appear almost black by transmitted light, owing to their opacity (see Fig. 10, *a*). These spheres are easily obtained by leaving a urine containing the amorphous urate to stand in the air until it becomes ammoniacal. The second form (*b*) occurs as very minute slender dumb-bells: these generally lie singly; or two lie athwart each other so as to form a cross; or three are united so as to form a rosette. They become coarser and larger with age. This deposit has no special *clinical significance;* its occurrence is merely an incident in ammoniacal decomposition of the urine. It is a frequent ingredient of the secondary phosphatic crust which invests urinary calculi in the later periods of their growth (see UROLITHIASIS).

V.—OXALATE OF LIME.

(*Oxaluria; oxalic acid diathesis.*)

Naked-eye characters.—A deposit of oxalate of lime is usually very scanty, and looks like a slight cloud of mucus. Owing to this, and its colourlessness, it seldom attracts the attention of a patient. If, however, the urine be transferred into a urine-glass immediately after emission, as is usually practised in hospital wards, the following appearances are produced, which are sufficiently characteristic to enable the observer to recognise the deposit with certainty by the unaided eye. The sides of the glass are seen to be traversed by numerous very fine lines, running in bands, transversely or obliquely, giving an appearance as if the glass were finely scratched. This appearance is due to the crystallisation of the oxalate on the fine lines or inequalities left after cleaning the glass by towelling. The subsided portion is equally peculiar; it consists of two parts —a soft pale-grey mucous-like sediment occupying the bottom of the vessel, and overlying this a snow-white denser layer with an undulating but sharply limited surface. The only other substance which crystallises in lines on the sides of the glass is uric acid: this is easily discriminated by the greater coarseness of the lines and their more or less brown colour.

Micro-chemical characters.—Oxalate of lime occurs in very minute crystals, the largest only appearing to the naked eye as sparkling points. Two forms are met with. The most common are octahedra, greatly shortened, or flattened, in one direction. The crystals present different appearances according to the side on which they lie. Commonly they rest on their short axis, and appear as squares crossed diagonally by a pair of lines (Fig. 11, *a*). As they roll over in the field of the microscope, they assume various forms—lengthened, pointed octahedra, crossed parallelograms, &c. (*b c*). Sometimes half-crystals are seen—four-sided pyramids on a square base (*d*); and sometimes two such pyramids, instead of being united by their bases to form the ordinary octahedron, are separated by a short square prism (*e*). The second form of oxalate of lime is that of dumb-bells and minute ovoids and circles (*f g*). The different appearances are produced by the different postures assumed by the objects; and as they roll over in the field of the microscope the dumb-bell is seen to change to an ovoid or circle, and *vice versâ*. Their real shape is that of an oval or circular disk, with rounded margins, and a depression in the centre on either face.

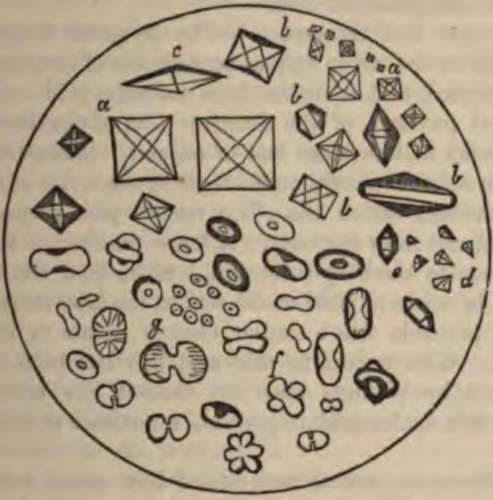

FIG. 11. Oxalate of lime—octahedra and dumb-bells, of various forms, and in various positions.

The dumb-bells are identical in composition with the octahedra. Dr. Bird, in his later editions, expressed a doubt on this point, on the ground of their different behaviour with polarized light. Dr. Thudichum, who investigated the matter afresh, has, however, shown that the octahedra have a polarising power equally with the dumb-bells, and that there is no reason to consider them as differing in chemical composition.

The precipitation of oxalate of lime as dumb-bells depends on some physical condition which interferes with the ordinary crystallisation. Very frequently urine depositing dumb-bells contains little masses of viscid mucus; and it seems probable that a certain viscidity of the urine is essential to this globular precipitation.*

Oxalate of lime is insoluble in alcohol, ether, water, and the vegetable acids; but it dissolves readily in the mineral acids. The urine depositing it is usually high-coloured and acid; very rarely neutral or faintly alkaline; and never, so far as I have seen, freely alkaline. Oxalate of lime is often conjoined with uric acid and the amorphous urates: much more rarely with the stellar phosphate of lime.

Production and occurrence. — The frequent occurrence of oxalic acid in the urine cannot be a matter of surprise when it is remembered that it differs from carbonic acid—one of the chief final products of the disintegration of the tissues—only in possessing half an atom less of oxygen. It constitutes probably one of the penultimate stages in the series of decompositions through which the effete tissues pass preparatory to their final exit from the body. A large number of substances which occur in the body (uric acid, creatin, fats, starch, sugar, &c.) can be made to yield oxalic acid in the laboratory; and it is highly probable that a similar change occurs in the living economy. With regard to uric acid this has been positively ascertained by Wöhler; and Dr. Garrod has succeeded in showing that oxalic acid is present, sometimes at least, in the blood.

It is therefore easy to understand how oxalic acid should exist in urine; also that it may be partly derived from the blood and appear in the urine at the moment of secretion, and partly be produced after the urine is secreted by conversion from uric acid. Dr. Owen Rees has nevertheless expressed his strong disbelief in the existence of oxalate of lime in the blood, apparently on the ground of the chemical difficulty in conceiving that oxalate of lime, from its insolubility, could exist dissolved in the blood; he contends that all the oxalate of lime found in urine is produced from uric acid after separation from the blood.* These theoretical objections, however, do not avail against the positive fact, that oxalic acid and its compounds, even the insoluble oxalate of lime, pass through the blood into the urine when introduced into the stomach. Wöhler found that oxalic acid given to dogs caused oxalate of lime to appear in the urine. Piotrowsky confirmed these results by experiments on himself. He took, in divided doses, from 80 to 100 grains of oxalic acid in the course of about six hours, and found that from 8 to 14 per cent. appeared in the urine as oxalate of lime, mixed with a little alkaline oxalate. Similar results were obtained with the oxalate of soda. When the insoluble oxalate of lime was taken in the same doses, very much less of it appeared in the urine; still, about 1½ per cent. could be recovered.†

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* The precipitation of carbonate of lime in spheres and close dumb-bells in the viscid urine of the horse is an example of the same kind. Mr. Rainey has shown a much wider application of the same principle in the calcifications which take place naturally in the hard tissues of the body. See Med.-Chir. Rev., vol. xx. p. 451.

Clinical significance.—Distinction must be made between slight occasional deposits, and large quantities occurring persistently. In the former case, it cannot be said positively that there is any departure from the normal state, seeing that oxalic acid is in all probability a natural constituent of urine: at least, it is constantly found in the urine of perfectly healthy individuals.

But when the deposit is *constant and large*, an abnormal state must be recognised to exist; and we are called upon to consider what pathological significance it may have, and whether it supplies any indications for treatment.

The most obvious inference is, that there exists a liability to the formation of an oxalate of lime calculus. This point, and the preventive treatment to be followed, will be considered in the section on calculous disease.

But a much wider significance has been given by some authors to oxalate of lime deposits: and a certain group of symptoms which are alleged to accompany these deposits, has been erected into a distinct pathological state under the name of the *oxalic acid diathesis*. Dr. Prout was the first to promulgate this view; and he has been followed by Dr. Bird and Dr. Begbie. Dr. Bird gives the following account of the symptoms which accompany oxaluria :—"They" (the patients) "are generally much emaciated, excepting in slight cases, extremely nervous, painfully susceptible to external impressions, often hypochondriacal to an extreme degree, and in very many cases labour under the impression that they are about to fall victims to consumption. They complain bitterly of incapability of exerting themselves, the slightest exertion bringing on fatigue. Some feverish excitement, with the palms of the hands and soles of the feet dry and parched, especially in the evening, is often present in severe cases. In temper they are irritable and excitable ; in men the sexual power is generally deficient and often absent. A severe and constant pain, or sense of weight across the loins is generally a prominent symptom, with, often, some amount of irritability of the bladder. The mental faculties are generally but slightly affected, loss of memory being sometimes more or less present." (Urinary Deposits, 5th ed. p. 251.)

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* "On Calculous Disease." Croonian Lectures for 1856, pp. 2 *et seq.*

† Archiv f. Physiol. Heilk., 1857, p. 122.

This train of symptoms is familiar enough to every practitioner ; and the occurrence of oxaluria in such cases is undoubtedly common enough ; but these symptoms may be present in typical completeness without oxaluria, and conversely oxaluria may exist in its highest intensity, and even go on to the formation of a mulberry calculus, without evoking any of the above-mentioned symptoms. Every one who has had experience in calculous disorders cannot have failed to observe that the subjects of mulberry calculus, especially children, are not unfrequently in the enjoyment of blooming health so long as no local irritation has been set up by the concretion. It will also not fail to be remarked that the symptoms attributed to oxaluria are almost identical with those attributed to spermatorrhœa. Disturbed equilibrium and loss of tone of the nervous system, with symptoms (more or less intense) of impaired digestion, are unfortunately a too common resultant of the intense activity of mind and body, and the trying wear and tear of modern life : and both physician and patient are naturally anxious to find some material alteration to account for a condition which is sufficiently serious, and which is remarkable for its want of definition. The patient often fixes on some derangement of the sexual function, generally, in these times, on spermatorrhœa, under the inspiration of unscrupulous publications too widely circulated among the curious public; or on heart disease, consumption, or gravel. The physician is able by means of physical examination to set aside these more open delusions, but falls himself into the trap of his own ingenuity, and is only more elaborately wrong than his patient. He finds crystals of oxalate of lime in the urine, and persuades himself that he has discovered the first link in the chain of consequences. It may be much questioned (and I certainly see no necessity for such a supposition) whether there be any morbid condition antecedent to the plain symptoms of the case, namely, a nervous system upset, because overtasked, and a digestion deranged, because mismanaged.

The facts and considerations which lead to the above reflections are:—

1. Intense oxaluria may exist persistently without evoking the group of symptoms attributed to the oxalic diathesis.

2. This group of symptoms may exist in typical development without the occurrence of deposits of oxalate of lime in the urine.

3. The most varied morbid states coexist with oxaluria. I have been in the habit for many years of noting the symptoms and pathological states of those patients in the Manchester Infirmary who had pronounced oxalate of lime deposits. Five out of every six exhibited none of those attributed to oxaluria. Almost every variety of disease was occasionally found associated therewith. The following especially were observed: chronic phthisis, cardiac affections, emphysema with chronic bronchitis, chronic rheumatism, anæmia, hemiplegia, malignant disease of the liver and stomach, chronic vomiting, and cirrhosis.

I am strongly convinced, that oxaluria arises from a variety of conditions, many of them not accompanied by appreciable departures from health, in which the assimilation of food or the disintegration of the tissues goes on imperfectly; and that it is impossible to assign any constant train of symptoms as the cause or the consequence of oxaluria. At the most, oxaluria is only one in a long list of symptoms, and one of the least significant.

Beneke, who has subjected this question to an elaborate examination, both in the way of experiment and observation, has formulated the following propositions, which appear to me to be well founded :—

1. Oxaluria, a condition which accompanies the lighter or severer forms of illness, has its proximate cause in an impeded metamorphosis—that is, in an insufficient activity of that stage of oxidation which changes oxalic acid into carbonic acid.

2. Oxalic acid has, if not its sole, its chief source in the azotised constituents of the blood and food : everything, therefore, which retards the metamorphosis of these constituents occasions oxaluria.

3. Such a retardation of the metamorphosis of the azotised constituents of the blood may be determined by the following causes :—

- *a.* Abuse of azotised articles of food (direct retardation).
- *b.* Abuse of saccharine and starchy articles of food (indirect retardation).
- *c.* Insufficiency of the red blood-corpuscles and (eventually) diminished oxidation.
- *d.* Insufficient enjoyment of pure, fresh, ventilated air.
- *e.* Organic lesions which in any way impede respiration and the circulation of the blood.
- *f.* Conditions of the nervous system which bear a character of depression, whether these arise primarily from mental derangement or from pathological states of the blood.

4. Excess of alkaline bases in the blood : which, as numerous observations tend to show, plays an important part among the ætiological conditions of oxaluria : and it is not improbable that an increased production of lactic and butyric acids in the digestive canal, consequent thereupon, impedes the development of the red blood-corpuscles, and thereby generates that chlorotic state which so often occasions and accompanies oxaluria.

5. Catarrhal conditions of the intestinal mucous mem-

brane, in case they are accompanied by oxaluria, have at most only a common source. They may determine oxaluria by causing deranged digestion, but cannot be considered as its proximate cause.*

Treatment.—After the foregoing reasoning and conclusions, it is scarcely necessary to say that oxaluria does not, in the opinion of the present writer, furnish special indications for treatment: nevertheless it will be found that, apart from the existence of organic disease, the conditions most frequently found associated with oxaluria, varied as they are, call for a tolerably uniform therapeutical action. They demand a quickening of the oxidation processes, and a careful regulation of the diet. The skin should be encouraged to activity by systematic use of cold sponging, friction of the skin with flesh-brushes, wearing of flannel vests and drawers, regulated exercise in the open air—if available, horse exercise. Many of the cases yield only to repeated change of air: the bracing atmosphere of upland and sea-side localities generally suits the best. It will often be found advantageous to withdraw for a time the use of tea and coffee, and to substitute milk: or if this prove heavy, milk mixed with one-fourth of lime water. The diet should be judiciously compounded of due proportions of animal and vegetable substances—diminishing the one or the other group of aliments according to the ascertained idiosyncrasy of the patient. He must be cautioned against heavy meals, and trained to partake more moderately of four meals a day. Digestion may be promoted by the administration of the mineral acids in light bitter infusions, or by small doses of the bicarbonate of potash in the same combination. It is not easy to determine beforehand which of these opposite medicaments will prove most grateful to the stomach. The rule of choice is, to administer the acid when the dyspeptic symptoms point to an atonic state of the organ and of the body generally; and the alkali when the signs point to gastric and general irritation.

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* Zur Entwicklungsgeschichte d. Oxalurie, by F. W. Beneke. Göttingen, 1852.

VI.—CYSTINE ($C_6H_6NO_4S_2$).

(Synonym—cystic oxide.)

Cystine or cystic oxide is a crystalline body of great rarity, which is found only under certain abnormal conditions in the bodies of animals. Hitherto it has been detected with certainty only in man and the dog. Cloetta asserts that he found it once in the kidneys of an ox.

Cystine was discovered by Wollaston in 1805, in a urinary calculus which was mainly composed of it. Since that time a considerable number of cystine calculi have been found in different parts of Europe and America; but, as compared with other urinary concretions, this is one of the most rare.

As a urinary deposit, cystine has been even less frequently met with than as a calculus; and as nothing is known touching the organic processes and constitutional states in which cystine is produced, the clinical interest attaching to it is for the most part confined to its manifestations as gravel and calculus. A number of cases have, however, been observed where cystine existed simply as a urinary deposit, or dissolved in the urine.

Generally, urine depositing cystine is turbid when voided: and on standing, a copious light sediment subsides, much resembling (to the naked eye) fawn-coloured lithates. The urine from which cystine is deposited has frequently a peculiar sweet-brier odour, a honey-yellow colour, and an oily appearance. It is usually faintly acid; and very liable to decomposition, in the course of which it evolves sulphuretted hydrogen, and blackens white glass vessels. Dr. Golding Bird observed that urine containing cystine changed from yellow to green when it became decomposed.

A few drops of acetic acid always precipitate an additional quantity of cystine from the supernatant urine; and if a urine containing cystine holds it all in solution, as may happen when the quantity is very small, acetic acid throws it down.

A deposit of cystine is not dissolved by heat, nor by the vegetable acids. It is instantly dissolved by caustic ammonia, and if the solution be exposed in a watch-glass to evaporation in the air, beautiful six-sided crystals are obtained as the volatile alkali exhales. This is the characteristic reaction of cystine, and leads to its easy identification. Cystine is also soluble in the carbonates of the fixed alkalies; but not in carbonate of ammonia, which indeed is its best precipitant from acid solutions. It is soluble also in the mineral acids, but insoluble in acetic and tartaric acids. It is insoluble in water and alcohol. Heated on a platina foil, it evolves thick white fumes, having a peculiar offensive odour resembling garlic.

Cystine is a body of very weak affinities, without taste or smell; it acts as a feeble base, and forms crystalline compounds with nitric and hydrochloric acids. According to Pelouze it may also play the part of an acid; he obtained two compounds with silver, which he denominated cystates.[*]

A spontaneous deposit of cystine in urine is composed of hexagonal tablets. When the ammoniacal solution of cystine is allowed to evaporate in the air, magnificent crystals are obtained, which furnish brilliant objects for the microscope. Cystine is dimorphous, and crystallises in two forms, namely, as six-sided tablets and square prisms (see Fig. 12).

The ammoniacal solution generally deposits hexagonal plates only, or these mixed with a few prisms; sometimes, however, the prisms are more abundant than the plates. The prisms either lie singly or form stars; they refract light strongly, and the facettes which lie slantingly out of the direct line of vision appear perfectly black, contrasting with the brilliant lustrous white of the planes through which the light passes vertically. This gives a peculiar striped appearance to the prisms, and causes them to appear deceptively six-sided. The hexagonal tablets have an iridescent mother-of-pearl lustre; their surfaces are often beautifully chased by lines of secondary crystallisation; they also form thick rosettes of great brilliancy.

The production of cystine in the animal body has as yet received no elucidation. It may, however, be assumed that, like other urinary ingredients, it pre-exists in the blood, and is merely separated by the kidneys. The most remarkable fact respecting the constitution of cystine is the large amount of sulphur (nearly 26 per cent.) which it contains. The close analogy of composition between it and taurine, renders it not improbable that the liver is the original source of cystine;* and the discovery of cystine in the livers of typhus patients by Scherer† lends support to this view.

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[*] "*Note sur la systine,*" by Pelouze, appended to Civiale's *Mémoire sur les calculs de cystine*, at p. 441 of Civiale's treatise *Du treatment médical de la pierre.*

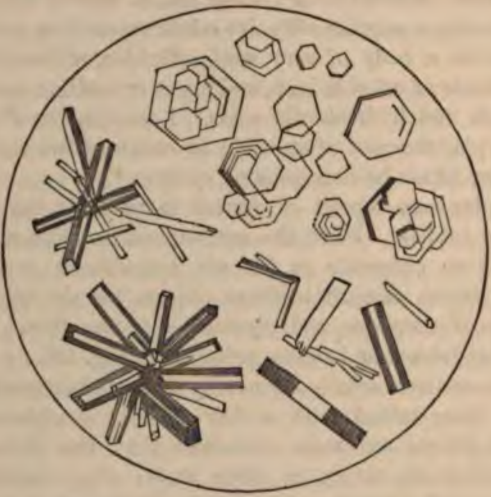

FIG. 12. Cystine. Hexagonal tablets and prisms.

The other constituents of the urine have not been found altered in any constant manner in cystinuria; and the later analyses of Beale‡ and Toel§ tend to support the original opinion of Civiale, that in cystinuria, as in most other calculous states, the composition of the urine, apart from the dominant

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* The close connection between cystine and taurine may be gathered at a glance by a comparison of their composition per cent.:—

	Cystine.	Taurine.
Carbon	30·00	19·20
Hydrogen	5·00	5·00
Nitrogen	11·66	11·20
Oxygen	26·66	38·40
Sulphur	26·66	25·60

† Archiv f. Path. Anat. Bd. x., p. 228.

‡ Urine and Urinary Calculi, p. 311.

§ Ann. der Chem. u. Pharm., Bd. xcvi., p. 24.

calculus-forming constituent, is normal. The excretion of sulphuric acid was not found diminished in a case examined by Beale. It would be of more interest to determine the amount of unoxidized sulphur voided with the urine in these cases. When it is remembered that from 3 to 5 grains of unoxidised sulphur are daily discharged with the urine by healthy men,* it would seem *à priori* not improbable that cystine is only the sulphur extractive in a new form. If it be so, one would expect the unoxidized sulphur to be diminished in cystinuria.

Deposits of cystine are very bulky; but the quantity, when weighed, is found unexpectedly small. Percentage determinations have been made by Prout and Beale. The former found 0·034, and the latter 0·09. Toel made a determination of the *daily* excretion in two sisters of Bremen, whose urine contained cystine both as a deposit and in solution. He obtained the same daily average in both—namely, 21·6 grains.†

Cystine may persist in the urine for many years; it may disappear for a while, and reappear again after a longer or shorter interval; or it may disappear permanently. It is sometimes succeeded by deposits of uric acid. The connection of cystine in the urine with deposits of the earthy phosphates, on which Prout and Civiale insist, is probably nothing more than a coincidence depending on the strong tendency of urine containing cystine to decompose and become ammoniacal, whereby the phosphates are necessarily precipitated.

One of the most curious circumstances in the history of cystine, is the unquestionable tendency which it shows to run in families. The facts bearing on this point will be referred to in treating of cystine calculus.

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* Ronalds: Phil. Trans. 1847, p. 461. The observations of Ronalds have been since confirmed by Griffith and Parkes. The same has been found in the urine of dogs by Bischoff and Voit.

† These results of Toel are, however, of doubtful trustworthiness, inasmuch as he seems to have followed a faulty analytical process. This was, first, to estimate the SO_3 in a known quantity of the urine; then to take an equal portion of the same urine, evaporate to dryness, and burn the residue with a mixture of carb. soda and nitrate of potash. He then determined the SO_3 in the fused mass; and having subtracted the sulphur previously found as SO_3, apportioned the rest to cystine. He seems to have forgotten that the urine might contain unoxidized sulphur, which would of course appear as SO_3 under the plan of analysis followed.

Cystine has been found mostly in children and young adults; though no age is exempt. Dr. Shearman, of Rotherham, believes that scrofulous children and chlorotic females are especially liable to cystinuria. In a young woman from whom Mr. Jordan extracted a cystine calculus some years ago, in the Manchester Infirmary, I found considerable tuberculous consolidation of both apices. The more recent researches of Fabre* do not support the opinions of Dr. Shearman. Fabre examined the urine of a large number of tuberculous persons and of thirty-six strumous children, but failed to detect a trace of cystine. In fifteen chlorotic females he likewise obtained negative results.

It is undoubted that persons may void cystine for years without any other deviation from health than what is caused by the physical irritation of the concretions, when these form. The brothers Planta, operated on by Civiale for immense cystine calculi, were known to have been excreting cystine in quantity for six years continuously, without any impairment of health. The sisters observed by Toel looked well, and were perfectly healthy, except that they were liable to nephritic pains from time to time, when they passed small calculi and gravel.

The *clinical significance* of cystine is therefore chiefly, if not wholly, the danger of the formation of stone and gravel.

The *treatment* of cystinuria, apart from that which is designed to prevent the formation of concretions, is necessarily, so long as the *rationale* of its production is so obscure, unsatisfactory. Dr. Prout believed he saw benefit from the long-continued use of nitro-muriatic acid. Dr. Bird, on the other hand, found the same remedy useless. If chlorosis or struma co-exist with cystinuria, these will of course demand their appropriate treatment; but as yet nothing is known which can pretend to have any direct influence in checking the formation of cystine. It may be borne in mind, however, that if the general health can be maintained at a good standard, and the danger of calculous formation warded off —both of which objects medical treatment may fairly hope to attain—the persistence of cystinuria may be looked on without much anxiety.

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* A. Fabre: *De la cystine*, &c. Paris Thesis, 1859. Fabre calls attention to the hexagonal appearance of uric acid crystals when precipitated by acetic acid; and he attributes the conclusions of Shearman to confounding these with cystine crystals.

VII.—XANTHINE ($C_{10}H_4N_4O_4$).

(*Synonyms—Xanthic oxide; uric oxide.*)

This rare substance was originally discovered by Dr. Marcet, about the year 1817, in a urinary calculus given to him by Dr. Babington. This concretion weighed only 8 grains, and had apparently been passed spontaneously. In 1816 the elder Langenbeck removed from a peasant boy, eight years of age, a stone as large as a small egg, which was afterwards identified by Stromeyer with the xanthic oxide or xanthine of Marcet. In 1837 a portion of this stone was analysed by Liebig and Wöhler;* in 1846 it was re-examined by Bodo Unger† with identical results. The name Xanthine was originally used by Unger to designate a substance found by him in guano, which he at first considered identical with Marcet's xanthic oxide, but which he subsequently established as a new substance under the name of guanine; the name xanthine then passed permanently to Marcet's xanthic oxide.

In 1829, Laugier‡ described some minute calculi obtained from a patient who had passed several. Three of these were handed over to Laugier; the largest of them weighed less than one-sixth of a grain. Their deep yellow colour, their spherical form, their smooth surface, seemed to indicate that they consisted of uric acid. They proved, however, to be xanthine, and yielded the characteristic reaction with nitric acid and potash.

Professor Dulk, of Königsberg, removed a xanthine calculus weighing 7 grains from the urethra of a boy (Bird).

Xanthine is a substance closely connected with uric acid, differing from it in composition only in possessing two atoms less of oxygen. Xanthine has been discovered by Scherer in the blood; also in the muscles, liver, spleen, and brain. Scherer[*] further states that a very minute quantity of xanthine is a natural constituent of healthy urine. Heller has been unable to convince himself of the correctness of this statement.[†]

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* Poggend. Ann. der Physik, 1837, Bd. xli. p. 393.

† Liebig's Ann. der. Chem. und Pharm., Bd. lviii., p. 18.

‡ Journ. de Chim. Méd., vol. v. 1st series.

Xanthine has been met with four times (as above recorded) as a urinary calculus: as a urinary deposit it is alleged to have been encountered by Bird, Douglas Maclagan, and Bence Jones. Maclagan found it mixed with earthy phosphates in the urine of a hysterical girl.[‡] Dr. Bence Jones's case was a school-boy between nine and ten years of age. Three years before he had suffered an attack resembling nephritic colic, but without subsequent passage of a stone. When first seen, the urine made at night contained a small quantity of albumen, but that of the morning contained none. A month later the urine was found "quite thick and deep coloured. A drop was placed under the microscope, and a crystalline deposit was found resembling one form of uric acid. From this form I considered the deposit was uric acid—(the crystals were pointed ovals). On examining the unfiltered urine for albumen by heat, I was surprised to see the crystalline deposit entirely dissolve. A fresh portion of sediment showed the same crystalline appearance and the same solubility by heat. . . . A day or two afterwards another specimen was brought to me, containing the same crystalline deposit soluble by heat. The sediment formed about an eighth of the bulk of the fluid. It was collected on a filter, washed with alcohol, and it gave the following reactions:—It dissolved in water and hydrochloric acid; when treated with nitric acid it dissolved without effervescence, and when evaporated to dryness it left a yellow residue."[§] Further examinations of the urine on subsequent occasions yielded no traces of xanthine.

Jackson thought he detected xanthine in diabetic urine;

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[*] Liebig's Ann. d. Ch. u. Ph., Bd. cvii., Heft 3, 1858.

[†] Heller's Harnconcretionen, p. 139, note.

[‡] Edin. Med. Journ., 1858, p. 121. Scherer doubts this case; and thinks the reaction mentioned by Maclagan insufficiently characteristic.

[§] Journ. of Chemical Society, Feb. 1862, p. 79. It may be remarked that in no previous account of xanthine have *crystals* of that substance been found. It is to be wished that in Dr. B. Jones's case the identification of xanthine had been more perfect.

but the tests he relied on were untrustworthy. Lehmann was unable to detect xanthine in several diabetic urines which he examined.

Purified xanthine, according to Städeler (who operated on xanthine obtained from Langenbeck's calculus), shows itself under the microscope as very small, irregular granules. When dried it forms brittle crusts, almost chalk-white, with a slight tinge of yellow, which become deeper-coloured when powdered. When rubbed, xanthine acquires a waxy lustre. It is soluble in alkalies; also moderately freely in concentrated and warm hydrochloric acid. This solution becomes turbid on cooling, and deposits quadratic octahedra of a combination of xanthine with the acid. It dissolves without effervescence in nitric acid, and the solution on evaporation leaves a bright yellow residue, which becomes violet-red when treated with solution of caustic potash.* The solubility of xanthine in water is subject to extraordinary variations, which are not yet understood. Städeler found pure xanthine from Langenbeck's calculus to dissolve in 13,333 parts of cold and in 1178 parts of hot water. Strecker found artificial xanthine, prepared by him from guanine, to vary in its solubility according as it was obtained from the evaporated ammoniacal solution or precipitated from its alkaline solutions by acetic acid. In the former case the solubility in hot water was, in round numbers, 1 in 1350; but in the latter, 1 in 396. Prolonged boiling was found by Strecker to lessen the solubility of xanthine in hot water.†

VIII.—LEUCINE AND TYROSINE.

These two substances were found by Städeler and Frerichs in the urine in typhoid fever and acute yellow atrophy of the liver. Tyrosine has even been found to form a natural urinary deposit in the latter disease. This deposit is described by Frerichs as a greenish-yellow crystalline sediment, which increases considerably with slight evaporation of the urine. Under the microscope, greenish-yellow globular masses, composed of acicular crystals are seen. In one of Frerichs' cases of acute yellow atrophy, he says of the urine:—"After standing in the cold air a greenish-yellow, light sediment was deposited, consisting entirely of acicular crystals of tyrosine aggregated together in globular masses. When a drop of urine was evaporated on a watch-glass, it left behind a residuum, which, upon microscopical examination, was found to be almost exclusively composed of the most characteristic possible crystals of leucine and tyrosine, partially saturated with colouring matter."* Frerichs regards the occurrence of these deposits as of great importance in the diagnosis of acute yellow atrophy of the liver.

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* Strecker: Liebig's Ann., May, 1861, Bd. cxviii., p. 158.

† *Ibid.*, p. 168.

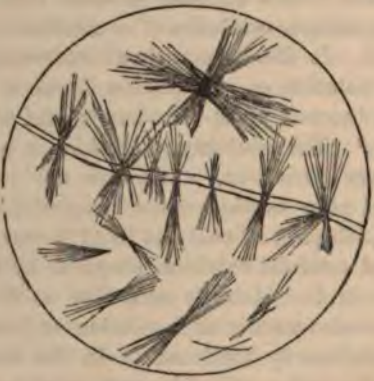

FIG. 13. Tyrosine, spontaneously deposited from the urine of a patient with acute yellow atrophy of the liver.

In May of the present year (1865), my assistant, Mr. Clements, brought me a specimen of urine passed by a young woman who was suffering (and died the day after) from acute yellow atrophy of the liver, in the home district of the Manchester Infirmary. After standing forty-eight hours, it had deposited an abundant sediment of tyrosine, crystallised in sheaf-like bundles of acicular crystals (see Fig. 13).

Xanthine, *hypoxanthine*, *guanine*, *tyrosine*, *leucine*, *creatine*, and *creatinine*, may be all regarded as intermediate steps in the regressive metamorphosis of azotised tissues, of which the ultimate stages are urea, uric acid, water, and carbonic acid. It is therefore not surprising that they should be found in small quantities in the tissues and the blood; and that a retardation of this metamorphosis in some particular stage should occasion their appearance in the urine.

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* Frerichs on Dis. of Liver, Syd. Soc. Trans., vol. i., Frontispiece, Fig. 5, and p. 220.

Hitherto their clinical significance has not been made out with sufficient clearness to be of practical service; and the circumstance that (except xanthine and tyrosine) they never form spontaneous urinary deposits, removes them (at present) from the interest and view of the practitioner. But it is not improbable that the study of these bodies in the urine may hereafter lead to important clinical indications; until then, it is not desirable to load a practical work like the present with details respecting them.

IX.—PHOSPHORIC ACID AND THE PHOSPHATES.

Phosphorus exists in the animal body in large quantities, either oxidized into phosphoric acid, and united with bases so as to form phosphates which pervade the fluids and solids—especially the bones; or unoxidized, and combined in some manner not yet understood with albuminous compounds.

Phosphoric acid passes out of the body partly with the fæces and partly with the urine. The diurnal excretion of phosphoric acid by the kidneys varies from 30 to 90 grains. The mean of twenty-five sets of observations collected by Dr. Parkes, was 48·80 grains a day. Two-thirds or three-fourths of this are combined with potash and soda to form soluble phosphates, which do not come under the notice of the practitioner as urinary deposits. The remainder is united with lime and magnesia to form salts, which, though soluble in acid urine, are speedily precipitated when the secretion becomes alkaline, and constitute urinary deposits.

Phosphoric acid is derived in part directly from the food; in part also from the oxidation within the body of the phosphorus of the albuminoid tissues, and especially the nervous tissue. The hourly excretion of phosphoric acid rises considerably after meals; and the earthy phosphates undergo a proportionally larger increase than the alkaline phosphates. In a series of observations extending over six days, I found that the average hourly separation of the earthy phosphates during the two hours preceding dinner, amounted only to one-half the quantity separated during the third and fourth hours after dinner. The alkaline phosphates rose from 3·47 grains per hour before dinner, to 4·90 grains after dinner.

The food is, however, not the sole source of the phosphoric acid of the urine; and the separation of it goes on, though in greatly diminished quantity, after prolonged fasting.

A very large number of observations have been made on the excretion of phosphoric acid in disease, but with results of slight clinical value. Dr. Bence Jones has formulated the following conclusions (founded on determinations per 1000 parts):—"In acute inflammation of the brain, there is an excessive amount of phosphates in the urine. When the inflammation becomes chronic, no excess of phosphates can be shown to exist. . . . . In some functional diseases of the brain, an excessive amount of phosphates is observable; this ceases with the delirium. Delirium tremens shows a remarkable deficiency in the amount of phosphates excreted, provided no food is taken. When food is taken the diminution is not apparent."* These observations are substantially borne out by the observations of Tomowitz and Beale.

Professor Vogel ascertained the rate of excretion of phosphoric acid in a very great number of acute and chronic diseases (having made above 1000 observations), but without eliciting any conclusions capable of clinical use.†

To the practitioner, therefore, the interest of phosphoric acid and the phosphates in the urine, is confined to the earthy phosphates which come before him as urinary deposits and urinary concretions.

Dr. Prout dignified with the name of "phosphatic diathesis," the tendency to the deposition of the earthy phosphates in the urine. Dr. Bence Jones‡ has, however, clearly shown that this designation is wholly inappropriate. There is not the least reason to believe that there is any constitutional state specially characterised by an excessive excretion of phosphates; the phosphatic diathesis of Prout is simply ammoniacal urine.

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* Medico-Chir. Trans., vol. xxxviii. p. 261.

† Neubauer and Vogel: Analysis of the Urine, Syd. Soc. Trans., p. 413—where the reader is referred for fuller information.

‡ Animal Chemistry, p. 85.

DEPOSITS OF EARTHY PHOSPHATES.

Phosphoric acid is spontaneously deposited in the urine chiefly, if not exclusively, in one of the three following combinations:—

1. Amorphous phosphate of lime, or bone-earth ($3\,CaO, PO_5$).
2. Crystallised phosphate of lime ($2\,CaO, HO, PO + 3\,Aq.$).
3. Ammoniaco-magnesian phosphate, or triple phosphate ($NH_4O, 2\,MgO, PO_5 + 12\,Aq.$).

These three compounds are occasionally precipitated together in one deposit; much more frequently the first and third are found together, forming the ordinary sediment of ammoniacal urine. This latter passes under various names, viz.:—"the mixed phosphates," the "secondary phosphates," or "fusible matter." This will come under notice again as the special constituent of secondary calculous formations.

The earthy phosphates are readily soluble in the natural acid of the urine; but are insoluble in neutral or alkaline fluids. They are consequently properly associated with an alkaline state of the urine; it is nevertheless a fact that the second and third occur occasionally in very feebly acid urines.

Urines depositing the earthy phosphates, or tending thereto, by their neutral or feebly acid reaction, usually become turbid when heated; but they clear instantly on the addition of any acid. This behaviour has been variously explained. Some have thought that the heat expelled the carbonic acid which held the earthy phosphates in solution; others, that the heat caused rapid decomposition of the urea into carbonate of ammonia, and thereby suddenly increased the alkalescence of the urine. Scherer thought the circumstance due to the conversion of the neutral phosphates of lime and magnesia into basic salts.

1.—Amorphous Phosphate of Lime, or Bone-earth.

This compound is invariably precipitated in alkaline urine. When the urine is alkaline from *fixed* alkali, this is the ordinary, and often the sole deposit; but far more frequently it is accompanied by the triple phosphate.

It forms an amorphous, whitish, light flocculent deposit, indistinguishable by the naked eye from epithelium. It has no affinity for the colouring matter of the urine, and is consequently of a paler colour than the supernatant urine, differing in this respect from the amorphous urates. The surface of the urine is generally covered with an iridescent film.

The application of heat does not dissolve the deposit, but, on the contrary, increases it. A drop of any acid causes it instantly to disappear. Under the microscope it appears as very pale, minute granules in irregular clumps or patches, much resembling the fawn-coloured lithates.

Its occurrence depends simply on the existence of an alkaline reaction, and the presence of lime and phosphoric acid in the urine.

This is the normal deposit of the alkaline urine after a meal. It is also frequently seen in persons whose urine has been rendered alkaline by remedies (carbonates, acetates, citrates, &c.), and after the excessive use of sweet and subacid fruits. The turbidity caused by the amorphous phosphate exists in its greatest intensity at the moment of emission of the urine, and does not increase on cooling.

The clinical significance and treatment of this deposit are entirely involved in those of alkaline urine. Bone-earth alone very rarely constitutes a urinary calculus; but it enters largely into the composition of phosphatic calculi in combination with the ammoniaco-magnesian phosphate.

2.—Crystallised Phosphate of Lime, or Stellar Phosphate.

Dr. Hassall first called attention to the existence of a crystallised form of phosphate of lime occurring as a urinary deposit. In 1860 he communicated a paper to the Royal Society on the composition and pathological importance of the calcareous phosphates occurring in the urine as a spontaneous deposit of stellar crystals. He considered these crystals to consist of biphosphate of lime; he also believed them of far graver significance than the triple phosphate of ammonia and magnesia.

In 1861 I had an opportunity of re-examining this question, and published the results of my observations in the British Medical Journal for March 30, 1861.

The crystals in question present considerable variety of form (Fig. 14).

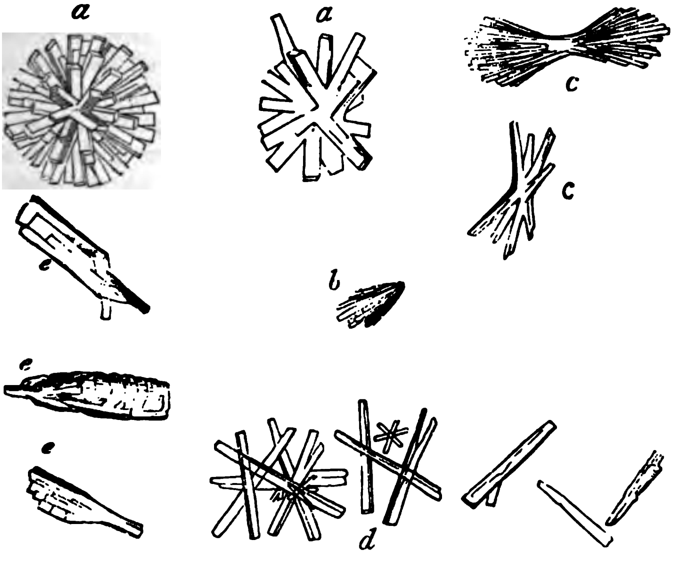

Fig. 14. Stars and rods of crystallised phosphate of lime, or stellar phosphate.

The prevailing appearance is that of crystalline rods or needles, either lying loose (*d*), or grouped into stars, rosettes (*a a*), fans (*b*), or sheaf-like bundles (*c*). Some of the crystals are club or bottle-shaped (*e e*), and abundantly marked with lines of secondary crystallisation.

In a case of diabetes under my care in the Manchester Infirmary, these crystals formed a constant deposit. The urine had been brought down by appropriate treatment to fifty ounces a day, and the patient was steadily gaining flesh and strength. The deposit was often mixed with oxalate of lime, and sometimes with uric acid; but never, except as the result of putrefactive decomposition, with the triple phosphate. I managed to collect about two grains of the crystals in a pure state, and subjected them to analysis. The results indicated the following formula: $2\,CaO, HO\,PO_5 + 3\,HO$.

By adding a little chloride of calcium to healthy urine, and reducing its acidity to near the neutral point with caustic soda, I have often succeeded in obtaining an abundance of crystals closely resembling those occurring spontaneously in urine. The reaction of the urine in which I have found the crystallised phosphate of lime has been generally faintly acid, often nearly neutral, and more rarely alkaline from fixed alkali.

The occurrence of a deposit of the stellar phosphate in urine is not common. It is, in fact, a rare deposit, as compared with oxalate of lime, uric acid, or the triple phosphate. The presence of this deposit in quantity is, according to my experience, an accompaniment of some grave disorder. In addition to the case of diabetes already mentioned, I have seen the stellar phosphates in cancer of the pylorus, once in phthisis, and more than once in patients exhausted by obstinate chronic rheumatism. They may, however, under peculiar conditions, be precipitated in a healthy urine. When the urine is rich in lime, and its acidity is at the same time depressed to near the neutral line, stellæ of phosphate of lime may form quite independently of any grave disorder, merely as the result of a coincidence in the chemical composition and reaction of the urine. For example, after a full meal, the acidity of the urine becomes greatly reduced, and lime derived from the food is in excessive proportion. In such circumstances, I have several times detected stellæ of phosphate of lime, but only in scanty numbers. A depressed acidity of the urine is an essential contingent to the formation of these crystals; and if the urine subsequently to their formation increase in acidity, they may spontaneously disappear.

3.—The Phosphate of Ammonia and Magnesia, or Triple Phosphate.

This is an insoluble crystalline compound, which occurs very frequently as a urinary deposit—sometimes alone, but much more commonly accompanied with the amorphous phosphate of lime. When unmixed with any other substance the deposit has a snow-white appearance; and bright, sparkling, colourless crystals are observed studding the sides of the urine-glass and forming a brilliant crystalline film on the top. The ordinary form of the crystals is a triangular prism with bevelled ends. A very great variety of subordinate forms are produced by a planing-off of the ridges and angles, and a hollowing out of the sides (Fig. 15). In a highly ammoniacal urine, the magnesian phosphate forms elegant di-elytral crystals, which appear to arise from a hollowing of the sides and a deep notching of the extremities of the prisms.

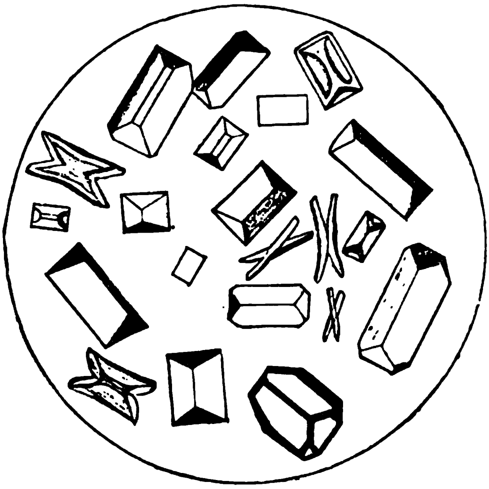

Fig. 15. Different forms of triple phosphate crystals.

The triple phosphate is easily soluble in acids; yet it may be found in urine that is feebly acid to test paper. Heat does not affect it; and the urine which deposits it commonly becomes turbid on boiling.

This deposit is necessarily present in ammoniacal urine, except in the very rare contingency of the urine not containing any magnesia. When urine is alkaline from fixed alkali, crystals of this salt generally appear after a while. This is easily explicable after the demonstration by Neubauer and Heintz that ammoniacal compounds exist in small quantities even in fresh natural urine.

In the immense majority of cases the deposition of this salt is only an incident due to the loss of the acid reaction of the urine, and especially of ammoniacal decomposition of the urine. Occasionally, however, it occurs in fresh urine which is neither decomposed nor sensibly (to the smell) ammoniacal. The following is the most remarkable instance which I have witnessed. J. P., a gentleman, aged twenty-nine, of a moderately healthy but irritable appearance, consulted me on account of a sense of weakness in the back and loins, with general debility and languor, and a tendency to sudden perspirations and fits of nervousness. There was severe smarting at the close of micturition. He had suffered from gonorrhœa three years previously, but had been completely free from any urethral discharge for some time. The urine was examined on several occasions. It was faintly acid when voided; and deposited, sometimes before it was cold, and generally within a couple of hours, an abundant precipitate of the unmixed ammoniaco-magnesian phosphate. The annexed note was taken of the urine voided at 11·30 a.m. on January 28, 1861. "In half-an-hour it was found transparent, perfectly sweet (*i.e.* not putrescent), faintly acid; and sparkling crystals of the triple phosphate could be seen floating in it. At four p.m. the same day, the specimen was quite clear; brilliant crystals of triple phosphate studded the sides of the glass, and at the bottom was collected an abundant snow-white deposit of the same crystals. The urine was not albuminous, neither did it contain pus or epithelium. On the following day the specimen continued unchanged; but on the fourth day the reaction had become faintly alkaline; the deposit was losing its snow-white character, and reddish flakes, composed of spheres of urate of ammonia, had become deposited. From this date the urine began to decompose, and speedily became ammoniacal and offensive." This condition of the urine, together with the unpleasant symptoms before noted, gradually disappeared in the course of six weeks, under the influence of cold sponging, systematic exercise in the open air, and the administration of dilute nitric acid in a bitter infusion.

X.—CARBONATE OF LIME.

When urine becomes alkaline from carbonate of ammonia, a small quantity of carbonate of lime is precipitated in an amorphous condition with the earthy phosphates. I have never seen it in a crystalline form in human urine: but it occasionally occurs in globular spheres and cornucopia-like crystals (Bird, Hassall). In the alkaline and viscid urine of the horse, carbonate of lime is frequently deposited in the form of minute spheres composed of radiating linear crystals, which are striking objects under the microscope. They show a dark cross with polarized light. The assumption of this globular form is probably connected with the viscidity of the urine.

Carbonate of lime constitutes a variety of urinary calculus which is of extreme rarity in the human subject, but much more common in the herbivora.

XI.—SULPHURIC ACID AND THE SULPHATES.

About 30 grains of sulphuric acid, in combination with alkaline bases, are daily excreted by the kidneys. A part is derived directly from the food, and a part from the oxidation of the sulphur contained in the albuminous compounds. The sulphates are highly soluble, and they never constitute a spontaneous urinary deposit.

In all febrile states sulphuric acid is increased. Dr. Parkes has observed a decided increase after the use of liquor potassæ. An increase is also observed after food, and in all conditions associated with an intensified metamorphosis of tissue.

It has not yet been shown that a knowledge of the quantity of sulphuric acid separated by the kidneys in any particular case of disease is capable of subserving any practical purpose.

XII.—CHLORINE AND THE CHLORIDES.

The chlorides never form spontaneous deposits in the urine; and the variations in their quantities have only an uncertain relation to special states of disease, but depend chiefly on the times of the meals and on the general rate of tissue-changes.

A good deal of attention has been called to the falling off or disappearance of the chlorides in the urine in acute pneumonia, and their reappearance when resolution is established. It has been asserted that a knowledge of the amount of chlorides excreted by the kidneys in the course of this disease, furnished valuable information for prognosis and treatment. Later observations have, however, shown that the indication is far from being a reliable one, and that the notions entertained in some quarters of its utility are greatly exaggerated. Although it be a rule of very prevalent application, that the chlorides in the increment stage of acute pneumonia are almost completely retained within the body, and that their reappearance in the urine is coincident with commencing resolution, yet there are exceptions to both these statements, especially to the coincidence of the reappearance of the chlorides with commencing defervescence (see Parkes).

SUPPLEMENTARY REMARKS ON THE EXCRETION OF PHOSPHORUS, SULPHUR, AND CHLORINE.

These three elements enter largely into the composition of the body, and they are abundantly present in articles of food. They pass out of the body chiefly with the urine; but partly also with the fæces. Multiplied observations have been made, and continue to be made, on the rate of their excretion both in health and disease; and important physiological and pathological deductions have been drawn from these investigations. It has been considered that the rate of excretion of phosphorus and sulphur, under proper precautions and corrections, furnished a measure of the exchange of material within the body—that is, of the activity of the molecular life of the tissues: and that in disease, an important insight into obscure phenomena could be thus obtained, capable of being turned to practical uses.

In proportion, however, as these researches have been extended, it has become clearer and clearer that these expectations are not likely to be realised, and that the practitioner is not likely to draw much help from these recondite sources. The difficulties in the way are manifold. In the first place, quantitative determinations of sulphur and phosphorus, notwithstanding all the aid of modern volumetrical methods, are still too troublesome and tedious to be within reach of any but a very select body of practitioners. But this is one of the smallest difficulties. In all such determinations it is necessary to do more than ascertain the proportion per cent. To obtain results of any value, the quantity per day must be ascertained. Again, there are physiological variations to make allowance for, arising from food, exercise, sleep, etc.; and, thirdly, it has now been ascertained that all known conditions remaining the same, the rate of excretion of these elements presents oscillations from an unexplained temporary retention, or partial retention, of the elements within the body, which is succeeded, after a shorter or longer interval, by a compensating increased discharge. These circumstances render it necessary to continue the observations over a number of days—six or eight—in order to cover the inequalities. For these and other reasons which might be mentioned, these inquiries are surrounded with difficulties. It is little wonderful, therefore, that the results obtained by different experimenters show a marked want of uniformity: and it is simply the fact that, from a clinical point of view, these laborious investigations must at present be regarded as infructuous, and for that reason they may be passed over with only a slight notice in a practical work. It is highly desirable, however, that researches of this class should be pushed on; it is impossible to say how soon practical lessons may be culled from these now apparently dormant facts. At any rate, they cannot fail to enlarge our general ideas on physiological and pathological processes.

XIII.—UREA ($C_2H_4N_2O_2$).

Looking at the urine from a physiological point of view, urea must be regarded as its most important constituent. It is the chief final product of the metamorphosis of the albuminous tissues, and furnishes the form under which nearly all the nitrogen finds its way out of the body.

Urea is a bland crystalline substance possessing the properties of a feeble base. Its best known combinations are the nitrate and oxalate, both of which are much less soluble than urea itself. Urea is very soluble both in water and alcohol; it never forms a spontaneous urinary deposit. Its presence in a urine of high density, or one artificially concentrated, is easily demonstrated. If to such a urine an equal volume of strong nitric acid be added, in a test-tube, and the tube be plunged into cold water, the mixture speedily becomes a shining mass of crystals of nitrate of urea.

The daily separation of urea by adult men between the ages of twenty and forty, averages about 500 grains; but the amount varies considerably from various causes, such as diet, exercise, meteorological conditions, and individual peculiarities. Of the twenty-four series of observations, of not less than six days each, tabulated by Dr. Parkes, the minimum result is 286·1 grains and the maximum 688·4 grains per day. The body weight has, as might have been expected, a very apparent relation to the daily excretion of urea, but the relation is not simply a direct one, because the weight of individuals is made up differently—some being heavy from bone and muscle, others from an accumulation of fat. It is estimated that a healthy adult man excretes urea at the daily rate of 3½ grains per pound of the weight of his body.

The excretion of urea is greatly increased after a meal—especially of animal food. Bidder and Schmidt believed that this arose from a direct transformation into urea of a portion of the alimentary materials without their being previously fixed as tissues: but Bischoff and Voit, with more probability, attribute this increase to an accelerated tissue-metamorphosis induced by the presence of the new supplies in the blood.

Copious water-drinking and exercise cause an increased separation of urea. Children secrete more in proportion to their weight than adults.

The quantitative estimation of urea in urine may be made by calculation from the specific gravity, or more exactly by the processes of Liebig and Davy. For general clinical purposes sufficiently accurate results may be obtained by the first method. The second and third methods are better suited for original researches.

(*a*). *Estimation of the urea from the specific gravity.*—Prof. Haughton has drawn up a useful table which indicates at a glance the quantity of urea excreted per day, provided the number of ounces of urine voided in the twenty-four hours, and the specific gravity of the collected quantity, be ascertained. The table, which is here somewhat abridged (see next page), is one of double entry, and the daily excretion of urea is obtained, in grains, at the points of intersection of the lines indicating the number of ounces, and the specific gravity of the urine.* This method is of course inapplicable to urines which contain sugar or albumen.

(*b*). *Liebig's Volumetrical method.*—This method is based on the property of urea to form an insoluble precipitate of fixed composition with the nitrate of the protoxide of mercury. But in order that the test may operate, it is necessary to free the urine beforehand from phosphates and sulphates. It is also necessary for complete accuracy to make allowance for the chloride of sodium present. When chloride of sodium coexists in any fluid with urea, the nitrate of mercury produces no precipitation of urea until the whole of the chloride of sodium is decomposed with formation of bichloride of mercury and nitrate of soda. After this conversion is completed, urea begins to be precipitated, and the test solution is to be added until no more urea remains in solution. This point is ascertained by a solution of carbonate of soda, which immediately develops a yellow colour when—and not before—all the urea has been thrown down with the mercury.

Three solutions are therefore required.

First.—A baryta solution, to precipitate the phosphates and sulphates. This is composed of one volume of a cold saturated solution of nitrate of baryta mixed with two volumes of saturated baryta-water.

Second.—The mercurial test solution. This contains 11·92

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* Med. Times and Gaz., Oct. 27, 1864.

PROF. HAUGHTON'S TABLE FOR THE ESTIMATION OF THE DAILY EXCRETION OF UREA FROM THE SPECIFIC GRAVITY.

Fluid Ounces.	Specific Gravity.																									
	1003.	1004.	1005.	1006.	1007.	1008.	1009.	1010.	1011.	1012.	1013.	1014.	1015.	1016	1017.	1018.	1019.	1020.	1021.	1022.	1023.	1024.	1025.	1026.	1027.	1028.
20	35	36	43	57	71	85	100	103	106	119	130	136	142	151	160	196	233	241	249	257	265	274	276	278	279	280
22	38	40	47	62	78	95	110	113	116	130	143	149	156	166	176	215	257	265	274	282	292	301	303	305	306	308
24	42	43	51	68	85	101	120	123	127	142	156	163	170	181	192	235	280	289	299	308	319	329	331	333	334	336
26	45	47	55	78	92	110	130	134	137	153	169	176	184	196	208	254	303	313	324	334	346	356	359	360	362	364
28	48	50	59	79	99	118	140	144	148	165	182	190	198	221	224	274	326	337	349	360	372	383	386	388	390	392
30	52	54	64	85	106	127	150	155	159	177	195	204	213	226	240	294	349	361	374	386	399	411	414	416	418	420
32	55	57	68	90	113	135	160	165	169	188	208	217	227	241	256	313	373	385	398	411	425	438	442	443	446	448
34	58	61	72	96	120	144	170	175	180	200	221	231	241	256	272	333	396	409	423	437	451	466	469	471	474	476
36	61	64	76	102	127	153	180	185	191	212	234	244	255	271	288	352	419	433	448	462	477	493	497	499	502	504
38	65	68	80	108	134	161	190	195	201	224	247	258	269	286	304	372	442	457	473	488	503	520	524	527	530	532
40	69	72	85	114	142	170	200	206	212	236	260	272	284	302	320	392	465	484	498	514	530	548	552	555	558	560
42	74	75	89	119	148	178	210	216	222	247	273	285	298	317	336	411	489	506	523	540	557	575	580	582	585	588
44	76	79	93	125	156	186	220	226	233	259	286	299	312	332	352	431	512	530	548	566	584	603	607	610	613	616
46	80	82	97	130	163	195	230	236	243	271	299	312	326	347	368	450	535	554	573	592	611	630	635	638	641	644
48	84	86	101	136	170	203	240	246	254	283	312	326	340	362	384	470	558	578	598	618	637	657	662	666	669	672
50	87	90	106	142	178	212	250	257	265	295	325	340	355	377	400	490	581	602	623	644	665	685	690	694	697	700
52	90	94	110	147	185	220	260	267	276	307	338	358	369	393	416	509	605	626	648	669	692	712	718	721	724	728
54	94	98	114	153	192	229	270	277	286	319	351	367	383	408	432	529	628	650	673	695	718	740	745	749	752	756
56	96	100	119	159	199	238	280	288	297	331	364	380	397	423	448	548	651	674	698	720	745	767	772	776	780	784
58	100	104	123	165	206	246	290	298	308	343	377	394	411	438	464	568	674	698	723	746	772	794	800	804	808	812
60	104	108	128	171	213	255	300	309	310	355	391	408	426	453	480	588	697	722	748	772	798	822	828	832	836	840
62	108	110	132	176	220	263	310	319	329	366	404	421	440	468	496	607	719	746	772	797	824	849	856	859	864	868
64	110	114	136	182	227	271	320	329	340	378	417	435	454	483	512	627	742	770	797	823	851	877	883	887	892	896
66	114	118	140	187	234	280	330	340	351	390	431	448	468	498	528	646	766	794	822	849	877	904	911	915	920	924
68	116	122	144	193	240	288	340	350	361	402	443	462	482	513	544	666	790	818	847	875	904	931	939	943	948	952
70	120	126	149	199	248	297	350	361	372	414	456	476	497	528	560	686	814	843	872	901	930	959	966	971	976	980
72	122	128	153	204	255	305	360	371	382	425	469	489	511	544	576	705	838	867	896	926	956	986	994	998	1004	1008
74	126	132	157	210	262	314	370	381	393	437	482	503	525	558	592	725	861	891	921	251	982	1014	1021	1026	1032	1036
76	130	136	161	216	269	323	280	391	404	449	495	516	539	573	608	745	884	915	946	977	1008	1041	1049	1054	1060	1064
78	134	140	165	222	276	331	390	401	414	461	508	530	553	588	624	765	907	939	971	1002	1034	1068	1076	1082	1088	1092
80	139	144	170	228	284	340	400	412	425	473	521	544	568	604	640	785	930	964	996	1028	1060	1096	1104	1110	1116	1120

grains (0·772 grammes) of proto-nitrate of mercury in ten cubic centimetres of water.

Third.—A solution of carbonate of soda of about the strength of twenty grains to the ounce.

As the preparation of the first and second solutions is very troublesome—the latter especially—it is more convenient to purchase them ready made.[*]

The analysis is performed in the following manner :—

1. Forty cubic centimetres (or two volumes) of the urine are mixed in a beaker with twenty cubic centimetres (or one volume) of the baryta solution. The mixture is thrown on a filter: fifteen cubic centimetres of the filtered fluid (which of course contain two-thirds, or ten cubic centimetres of urine) are carefully measured off and placed in a small beaker.

2. A graduated burette is filled with the mercurial solution, which is then very carefully dropped into the beaker until the mixture begins to become turbid; a few drops generally suffice. A note is taken of the quantity of the solution used to reach this point : it indicates that all the chloride of sodium is decomposed and that the urea is now beginning to be precipitated.

3. The mercurial solution is now added more freely, and thoroughly mixed by means of a glass rod : a copious white precipitate makes its appearance and the analysis approaches completion.

4. This point is ascertained by pouring some of the carbonate of soda solution into the bottom of a white porcelain plate : and taking a drop from the turbid mixture in the beaker by means of the stirring-rod, and letting it fall into the solution on the plate. As long as the drop produces only a white curdy circle the mercurial solution is to be still added ; but as soon as a yellow tinge appears the analysis is finished.

5. The quantity of mercurial solution used is then read off, and the portion used before the occurrence of turbidity subtracted—the remainder is what has been employed to precipitate the urea. Each cubic centimetre of the solution used indicates

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[*] These and other test solutions for volumetrical analyses of the urine may be had of Griffin, Bunhill Row, London ; and from Siebold, Chemist, Oxford Road, Manchester.

0·154 grain (or 0·01 gramme) of urea. From this, by an easy calculation, the amount of urea in ten cubic centimetres of urine may be ascertained; and if the number of cubic centimetres of urine voided in the twenty-four hours be known, the daily excretion of urea is readily calculated.

(*c*). *Davy's process* is, on the whole, less convenient and exact than that of Liebig; it will be sufficient to indicate the principle of it. When a solution of hypochlorite of soda (liquor sodæ chlorinatæ) is added to a solution of urea, the latter is speedily decomposed, and its nitrogen set free in the gaseous state. The volume of nitrogen evolved furnishes an exact measure of the urea decomposed—each cubic inch of the gas corresponding to 0·65 grain of urea.*

Pathological relations of urea.—The excretion of urea in disease has been examined in a large number of cases. In the acute stage of febrile and inflammatory diseases, there is an increased formation and discharge of urea, depending on an accelerated metamorphosis of tissue. When the crisis of the disorder has passed, and defervescence sets in, the excretion of urea falls even below its natural average. This rule, however, is liable to exceptions; it appears that in not a few instances there is a retention of urea within the body during the pyrexial period, even when no disorder of the kidneys exists, and a compensating discharge when convalescence begins. Acute (inflammatory) Bright's disease is a constant exception: the urine in that complaint is poor in urea: but this arises not from diminished formation, but from defective separation, owing to the blocked-up condition of the uriniferous tubes.

Frerichs found in one example of acute yellow atrophy of the liver, a total deficiency of urea in the urine: in a second case there was abundance of urea in the urine discharged during life, but only a trace in that withdrawn from the bladder after death.

In chronic diseases not involving the kidneys, the excretion of urea has not usually been found materially affected.

In saccharine diabetes there is an excessive separation of urea, as might have been expected from the accelerated rate of tissue-metamorphosis, which must accompany the full feeding and rapid emaciation of these patients.

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* For fuller details of this analysis I must refer to Davy's paper in the Philos. Mag., June, 1854; also to Dr Thudichum's Treatise on the Pathology of the Urine, where this method, and that of Liebig, are described at length.

In a case of diabetes insipidus (with a daily discharge of 12 or 14 pints of urine), I found the excretion of urea to oscillate between 394 and 505 grains daily, which yielded a mean rate of 4½ grains per pound of the body weight. This is about a fourth above the average for healthy individuals.

In both acute and chronic degeneration of the kidneys (Bright's disease) there is a marked lessening of the excretion of urea, as will be more fully commented on when those diseases come to be described.

One of the most important properties of urea is the great facility with which it is broken up and resolved into new compounds. This property comes into important play when urea is unnaturally retained in the blood or in the urinary passages. It has been already explained with what consequences this bland and innocuous base is converted into pungent carbonate of ammonia in the bladder and other parts of the urinary tract. A similar conversion, taking place in the blood, is the cause, according to Frerichs, of the stormy and dangerous phenomena of uræmia.

Dr. Prout believed that there existed a peculiar morbid state characterized by an absolute and relative increase of the excretion of urea, unaccompanied by pyrexia. To this condition Dr. Willis, who adopted the view of Prout, gave the name of *Azoturia*. The subjects of this form of disease, according to Prout, had usually a frequent and urgent desire to pass water both by night and day. This seemed principally due to an irritable sensation referred to the neck of the bladder, occasionally extending along the urethra; but in some cases it was due, at least in part, to real diuresis. In almost every instance the quantity of urine voided in the twenty-four hours was somewhat above the natural standard. The quantity was also particularly liable to be increased by causes which would scarcely affect a person in perfect health, at least to the same degree; such as by a chilly state, mental emotion or excitement, etc.*

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* Prout: Stomach and Renal Diseases, 5th edit., p. 97.

In addition to the direct urinary symptoms, there was sometimes a sense of weight or dull pain in the back, accompanied by disinclination to bodily exertion; there was no remarkable thirst; nor craving for food; nor emaciation. Moreover, the functions of the skin appeared to be little deranged.

Such is a summary of the description of Prout. He does not supply any details as to the daily flow of urine nor the daily amount of urea. At the time Prout wrote, very little was known as to the natural (physiological) variations in the excretion of urea; and the opinion he held as to urea being chiefly the final product of the metamorphosis of the gelatinous tissues has since been proved to be erroneous. Looking at the question from the standing-point of the physiological doctrines now in the ascendant, it is difficult to admit the existence of a condition characterized by the incompatible coincidences of an increased excretion of urea, with absence of thirst, absence of excessive feeding, and absence of emaciation.

Precise facts in support of Prout's view are wanting. Willis's description is too loose to give much confidence, and subsequent writers have contented themselves with a reference to Prout and Willis.*

Dr. Parkes,† however, records a remarkable case examined by Dr. Ringer. The patient was a middle-aged man weighing 109 pounds, who was not febrile, and appeared only feeble. He was fed on the ordinary diet of the hospital (University College), and passed in each twenty-four hours no less than 1130 grains of urea (mean of twelve days), or 10·36 grains to each pound avoirdupois of his body-weight. There was a trace of sugar, but not enough to determine quantitatively. The daily flow of urine in this case amounted to 96 fluid ounces, which is fully double the normal average.

In my own experience, I have usually found that cases which at first sight appeared to belong to this category—cases exhibiting a dense urine and a train of nervous symptoms—turned out on more exact investigation to want the special feature indicated by Prout as the essential one; namely, an absolute increase in the daily discharge of urea. Nevertheless, some facts, rarely observed, have left an impression on my mind that Prout's description is not altogether fanciful. The following case, which I saw with Mr. Greaves of this town, seems to have been one of those Prout had in view when he drew up his account.

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* The six cases recorded by Dr. Handfield Jones in the Brit. Med. Jour. for Oct. 12, 1861, under the title of "Cases of Baruria," are so deficient in necessary details that they are of no service to a reader. In only one of them was the urine of the twenty-four hours collected and examined, and in that case only on one occasion. In the remainder "baruria" seems to have been inferred to exist from the high density of a single specimen.

† Parkes: On the Composition of the Urine, p. 374.

Mr. L., a man about 50, complained of troublesome irritation at the back of the pharynx, debility, want of energy and power of application to business. In the preceding three months he had lost 20lbs. in weight.

The urine was first examined by me on May 23, 1863. It had sp. gr. 1029, and contained a small quantity of sugar, but less than one grain to the fluid ounce. This was the only occasion on which I detected sugar, but Mr. Greaves had found it once or twice previously. It was arranged that the whole of the urine voided in each 24 hours should be separately collected and sent for analysis. This was done for three successive days; and three weeks later it was done again for two successive days. The following table exhibits the result of the examination :—

		Quantity per day.	Sp. gr.	Total urea.
May	25 .	. 27 ounces	1029·5	542 grains.
,,	26 .	. 30½ ,,	1029·75	559 ,,
,,	27 .	. 31 ,,	1028·25	555 ,,
June	18 .	. 29 ,,	1027·5	565 ,,
,,	19 .	. 34 ,,	1020·5	510 ,,

This patient was not febrile; his weight was 120 lbs.; there was little appetite, and no thirst, and yet he excreted daily 4·6 grains of urea for each pound of body-weight on these five days, which is fully a quarter beyond the usual average. I saw the patient again toward the end of January, 1863. The urine had then lost its peculiarity; and the health, under a regulated diet and exercise, and a course of vegetable tonics, with citrate of potash, had become completely re-established.

Prout was of opinion that these cases were pathologically related to diabetes; and he conjectured, though he had not witnessed the fact, that they often developed subsequently into that disease. That there is some relation between the two conditions seems not improbable; in the cases of Dr. Ringer and myself a small quantity of sugar was temporarily present in the urine with the excess of urea.

In the case just related the cause of the complaint was mental anxiety; and in all the instances which I have been inclined to place in this group, the origin of the disorder could always be traced to some kind of mental emotion.

CHAPTER IV.

ABNORMAL SUBSTANCES IN THE URINE: ORGANIC DEPOSITS.

I.—PIGMENTARY PARTICLES.

THESE objects deserve a passing notice, from the fact that they are frequent, almost constant, if not absolutely constant, objects in urinary deposits, and have not hitherto been described. They are especially abundant when there is a copious shedding of epithelium into the urine; but they are not associated with any particular kinds of epithelium, and appear indiscriminately with renal, vesical, urethral and even vaginal epithelium.

They never exist in such quantity as to form the entire of a visible urinary sediment; they are only to be recognised by the microscope. They vary exceedingly in size, shape and colour: and yet they are easily identified. They appear especially under two conditions: namely, as free amorphous particles, and cell-like bodies (or celloids). The accompanying figure represents the more common forms (Fig. 16).

The free amorphous masses (*a a*) are of a reddish-brown or brownish-orange colour, varying in size from mere specks just visible under the microscope to particles as large as pus globules or larger.

The cell-like particles have a peculiar appearance, very difficult to explain. They never present an unmistakeably cellular character; they appear flat, never spherical. Their outline is generally an oblique ovoid. Within this outline, which is generally of exceeding delicacy, and of perfect definition, lie masses of red or orange pigment exactly resembling the free amorphous particles already described. Sometimes the pigment fills the entire outline (*c*); but generally some portion of the celloid is free, and the pigment is accumulated in one or more heaps filling up the outline to very different degrees (*b b*). Sometimes there is only just a granule of pigment in the entire celloid, or there may not even be any in at all (*d*). In the last case the celloid has a peculiar, faint, blueish, mother-of-pearl, lustre. When the celloid, on the other hand, is stuffed, as it were, with the pigment, it loses its transparency and appears almost black in the field of the microscope (*e*). Not unfrequently the outline is incomplete, presenting an appearance as if half or more of it had been carried away, but without at all interfering with the condition of the remainder (*f*).

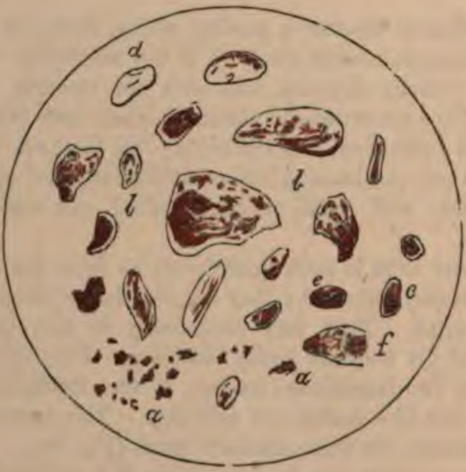

FIG. 16. Pigmentary particles. *a a*. Free amorphous particles; *b b*. Celloids, containing pigmentary particles.

The celloids vary in size from a mere speck, less than a blood corpuscle, to a superficies as large as that of a pavement-epithelium cell. The ordinary dimensions are from $\frac{1}{1500}$ to $\frac{1}{1000}$ of an inch. They keep badly in urine, and disappear altogether when decomposition sets in.

These particles are not confined to any particular state of the urine, and have not, so far as I have ascertained, any precise pathological signification; though they are always much more numerous in Bright's disease, vesical calculi, purulent urine, leucorrhœa and urethritis, than when the urine is free from morphological elements.

I am unable to explain their presence in urine. I have found them in quantities in the atrophied kidneys of chronic Bright's disease. But I have also found exactly similar bodies in places far removed from the urinary passages—in the brain, in the vicinity of old apoplectic clots, in the neighbourhood of old extravasations in other places, in encephaloid growths and other tumours.

I have been in the habit of noticing these objects for many years, and have regarded them as derivatives of hæmatine; but how they come to assume their peculiar forms I cannot conjecture.

II.—EXTRA-RENAL EPITHELIUM.

Any part of the genito-urinary passages may shed its epithelium into the urine so as to form a sediment.

The urines of the two sexes differ notably in the character and quantity of the epithelial cells found in them. This arises from anatomical differences in the lower genito-urinary passages; and advantage may sometimes be taken of this circumstance to distinguish the sex of the individual whose urine is under examination.

In the male sex an epithelial deposit of extra-renal source is most commonly derived from the urethra and prostate gland, and is composed of oval, tailed or rounded cells (Fig. 17), about twice as large as pus cells and usually flattened. A deposit of this sort is always scanty, and to the naked eye presents the appearance of a collection of whitish flakes and strings. When taken up by the pipette for examination, these flakes are found to have the viscid glairy character of mucus. A sediment of this character is not uncommon in men; in many cases it may be distinctly traced to an old gonorrhœa, which has long since passed away leaving no other vestiges behind it. The deposits found in the urine of persons subject to nocturnal emissions have very much the same appearance to the naked eye.

It is well to be aware of the nature of this deposit. Youths principally, but older men not unfrequently, observe for themselves the presence in their urine of the strings and flakes just described; and they are liable to become subject to hypochondriacal fears and anxiety respecting them. Such individuals are common victims of unprincipled empirics. I was recently consulted by a gentleman who paid very large sums to a quack who had persuaded him that the flaky shreds in his urine—the innocuous vestiges of a gonorrhœa contracted five years previously—were of a dangerous nature, and required active and long continued treatment. It is not a trifling matter to be able to allay the alarm of such patients, and to convince them that the subject of their anxiety is wholly unimportant.

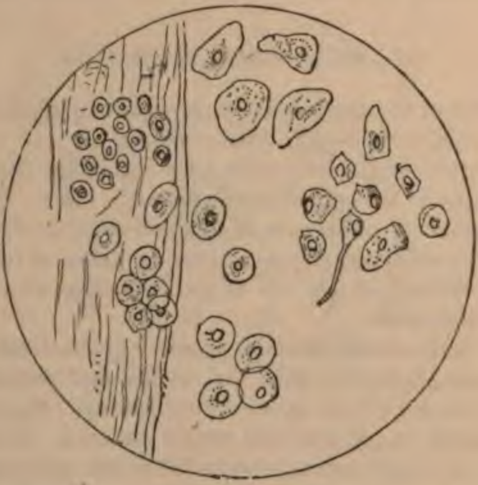

FIG. 17. Oval and tailed epithelial cells, found in the thready and flaky deposit of the urine of a man who had formerly suffered from gonorrhœa.

In females, epithelial sediments are both common and abundant. From the simple short urethra the urine receives little or nothing; but the vaginal membrane is invested in an extensive tract of pavement epithelium, the elements of which are detached with facility and in great quantity, giving rise to an abundant amorphous-looking, light, cloudy deposit in the urine. When examined microscopically this deposit is found composed of large flat cells resembling the epithelia of the mouth

(Fig. 18). The cells either lie discrete, or united by their borders into patches of rude mosaic.

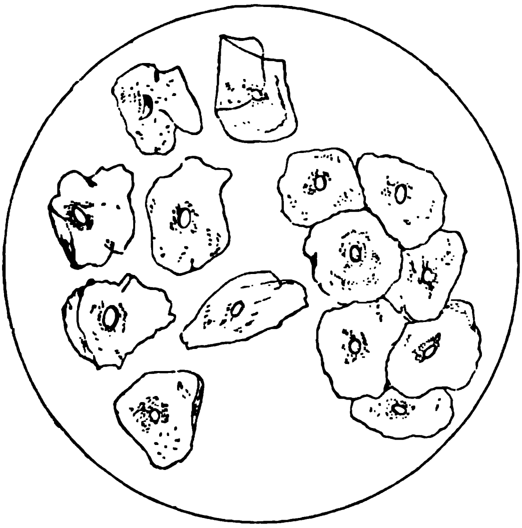

FIG. 18. Vaginal epithelium in the urine.

A deposit of this character is found only in the urine of females, and comparatively few are wholly exempt from it. In the subjects of vaginal leucorrhœa it is always abundant; but it is also present frequently, and in quantity, where there is no appreciable disorder of the genital passages. Even young (female) children may have a sedimentary urine from this cause, especially those of a strumous habit.

The epithelium of the bladder, ureter and pelvis of the kidney finds its way into the urine of both sexes in cases of vesical calculus, renal calculus, and pyelitis from any cause. The epithelium which lines these parts is of a transitional character, and presents a great variety of forms—cylindrical, spindle-shaped, caudate, oval, spheroidal and irregular (Fig. 19). It cannot fail to be noticed how like some of these cells are to cancer-cells: so like indeed that the recognition of cancer-cells (as such) in the urine becomes a matter of very great uncertainty. In cases of suspected pyelitis the existence of cells of this class in the urine, greatly fortifies the diagnosis. (See PYELITIS.)

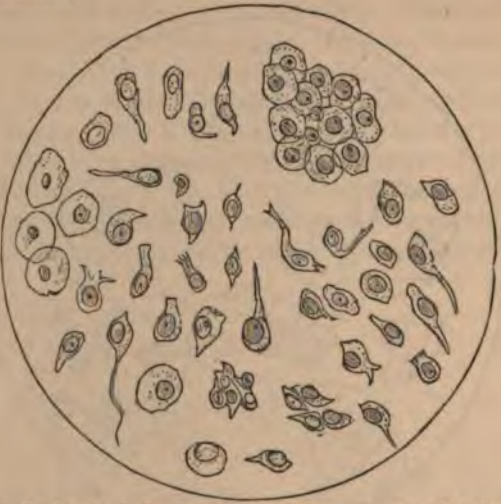

FIG. 19. Epithelial cells from the bladder, ureter, and pelvis of the kidney.

III.—RENAL EPITHELIUM AND CASTS OF TUBES: THE DEPOSITS ASSOCIATED WITH ALBUMINURIA.

As renal epithelium, and casts of the uriniferous tubes, are commonly found together, it will be convenient to consider them in conjunction.

The uriniferous tubes are lined with a single layer of spheroidal epithelium. The cells of this layer in the cortical part of the kidney consist, in the healthy state, of a round or slightly oval nucleus having a delicate, regular outline, resembling closely, both in size and aspect, (except in not being biconcave) the red corpuscle of the blood; around this nucleus is aggregated a quantity of solid, yet friable, faintly granular substance (Fig. 20 *a a*). A distinct cell-wall is usually understood to exist around each nucleated mass; but my own observations tend to support the view of Dr. Beale that, in the convoluted tubes, a distinct cell-wall can only occasionally be seen. When the cut surface of a healthy kidney is scraped, the nucleated masses are freely separated from each other. The nucleus itself is then seen to be exceedingly uniform in size and shape; but the granular matter surrounding it is very irregular. Sometimes the nucleus is quite free; more commonly it is embedded in granular matter. Sometimes this latter forms a spheroidal mass, with a more or less distinct cell-wall; sometimes the granular mass looks as if partly broken off; or there remains only a small quantity of it adhering to the nucleus. In the straight tubes the cell-wall is always distinct enough, and the cells are flatter, so that the available bore of the tubes is larger in the pyramidal than in the cortical portions. When there is rapid proliferation of the epithelium of the uriniferous tubes—(as in the large white or mottled kidney) the nucleus is frequently seen cleft into two or three nucleoli, and the cell puts on the appearance of a pus corpuscle.

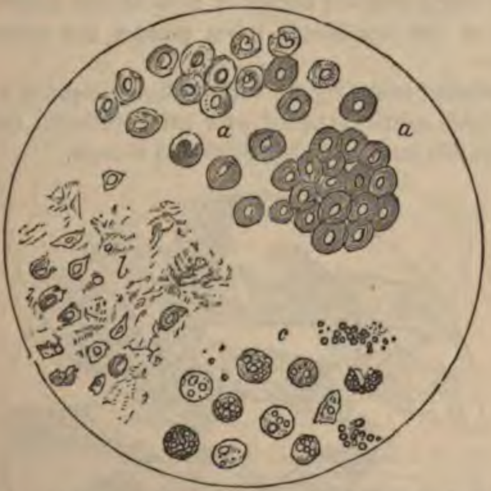

FIG. 20. Renal epithelium. *a a*. Natural appearance of the cells; *b*. Atrophied and disintegrated renal cells; *c*. Renal cells in a state of fatty degeneration.

The epithelial lining of the uriniferous tubes is liable to be separated from the basement membrane, in certain diseased conditions, and discharged with the urine. Coagulable matter is also liable to be poured into the uriniferous tubes, and having solidified there, is afterwards washed out by the stream of urine, and appears therein as casts or moulds of the tubes.

The epithelium and casts thus discharged, present a number of modifications of form and aggregation which serve to indicate and distinguish particular states of disease in the kidneys.

Renal epithelium, forming a urinary deposit, occurs usually in scattered patches; but in the acute form of Bright's disease the epithelium is detached in coherent pieces which constitute casts of the entire lumen of the tubes. These are "the epithelial casts" to be presently mentioned. The cells never perhaps preserve perfectly their normal form. The most common change is a greater or less disintegration or breaking-up of the cells into amorphous granular matter, which readily takes place from the absence or extreme tenuity of the cell-wall. The cells also become atrophied (Fig. 20 *b*); and not unfrequently degenerated into fatty corpuscles (*c*) which are significant of changes in the kidney of most serious nature.

Casts of the uriniferous tubes present the following varieties:—

1. *Epithelial casts* (Fig. 21 *a a*).—These consist of a cylinder of coagulable matter, studded over with epithelial cells which adhere thereto and are partly imbedded therein.

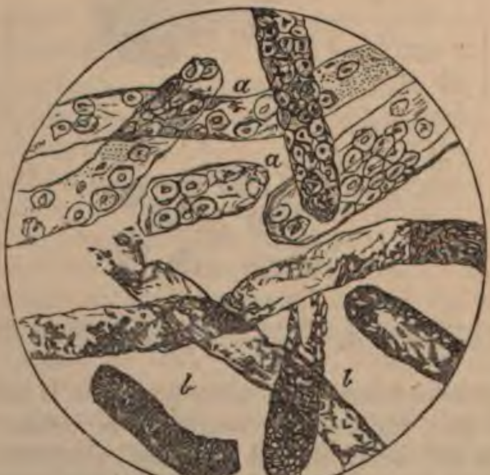

FIG. 21. *a a.* "Epithelial" casts; *b b.* "Opaque granular" casts, from a case of acute Bright's disease.

2. *Opaque granular casts* (Fig. 21 *b b*).—These have a dark coarsely granular appearance, and are generally of medium size ($\frac{1}{700}$ of an inch in diameter).

3. *Transparent or waxy casts* (Fig. 22).—These are clear, glassy, fibrinous cylinders, sometimes so transparent as to be invisible until tinted artificially by means of Iodine or a solution of Magenta; sometimes faint markings map their surface, or they show a faint molecular composition. They present extreme differences of diameter; the smallest are not more than the breadth of a blood corpuscle (*a a*); the largest are $\frac{1}{300}$ of an inch, or more, in breadth (*b b c*); others again are medium sized.

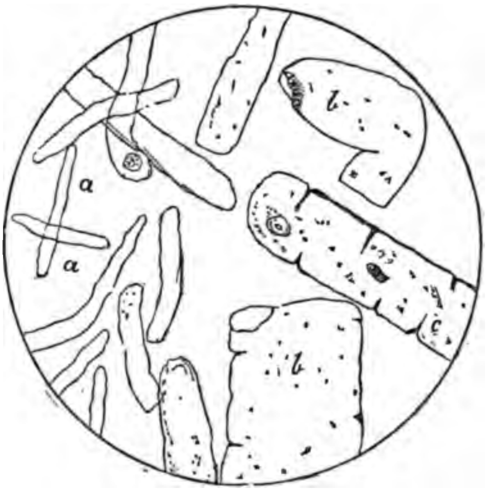

FIG. 22. Waxy casts. *a a*. From the urine of a man with chronic Bright's disease of eight months' duration (urine bloody, intensely albuminous, anasarca, death from pneumonia); *b b*. From a case of chronic Bright's disease (large white kidney); *c*. From a case of chronic Bright's disease (contracted kidney with fatty degeneration).

4. *Fatty casts* (Fig. 23 *a a*).—Sometimes a transparent cast is studded with tolerable uniformity with minute oil particles: more commonly the oily particles are irregularly distributed in and on such a cast; sometimes again they are collected into dark botryoidal masses—apparently the result of the breaking-up of an adherent cell which has undergone fatty degeneration.

5. *Blood Casts* (Fig. 23 *b b*).—Sometimes these are exceedingly beautiful objects, being perfect cylinders composed of delicate circles placed in apposition ; more generally a fibrinous cast is studded irregularly with blood corpuscles, some perfect, and some withered and contorted ; sometimes the cast seems composed of blood disks crushed or compressed into a cylindrical mould (*c*).

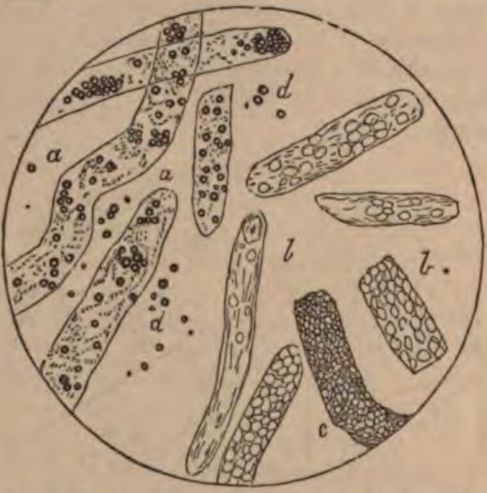

FIG. 23. *a a.* Fatty casts ; *b b.* Blood casts ; *d d.* Free fatty molecules.

6. *Pus Casts.*—Dr. G. Johnson has described and figured moulds or casts of the uriniferous tubes composed of pus corpuscles. In two such cases, examined *post mortem*, he found multiple abscesses in the kidneys. In a case examined by myself, where both kidneys were riddled with myriads of secondary abscesses, the urine found in the bladder after death contained no recognisable tube-casts; the observation was, however, an imperfect one, owing to commencing ammoniacal decomposition of the urine, which may have caused disintegration of any pre-existing casts.

To the naked eye deposits of renal epithelium and tube-casts appear amorphous: they are often very scanty, and resemble a cloud of mucus: sometimes they are more dense and form a white flour-like sediment.

Clinical significance of renal epithelium and tube-casts.—The most universal inference from the presence of these bodies in the urine, is the existence of some organic mischief in the kidneys. But a study of their various forms and appearances furnishes still further information of great weight in the diagnosis and prognosis of the different stages, and different types of renal degeneration. This subject cannot, however, be advantageously considered in the present section, but will take its place more appropriately in the chapters on Bright's disease. The following general remarks may, however, find room here :—

1. The deposit may, and generally does, contain a mixture of two or more varieties of casts and cells.

2. Conclusions as to their pathological meaning must be deduced from the prevailing types rather than from the absence or presence of one or two of a particular character. For example, it must not be assumed that the kidneys are in a state of hopeless fatty degeneration, or even commencing to undergo that change, because one or two cells, or one or two casts display oil molecules.

3. It is necessary, in order to avoid serious errors, to examine specimens of urine passed on two or three separate days.

4. Bearing in mind these precautions, and having regard to the previous history of the case, the following conclusions are *generally* warranted. (*a*). Epithelial casts and blood-casts indicate a disease of recent origin. (*b*). Transparent large waxy casts, mixed with dark granular casts, indicate a chronic disease. (*c*). Epithelium and casts containing much fat indicate fatty degeneration.

IV.—FATTY MATTER IN URINE.

Fatty matter appears in urine under a variety of circumstances.

1. In the preceding section it has been shown that tube-casts and renal epithelium (sometimes vaginal epithelium also) are liable to undergo fatty degeneration, and oily particles then appear in the urine, either enclosed in the altered cells or lying free. Dr. Beale has shown that the oily matter under these circumstances contains cholesterin dissolved in it.

2. In the condition called chylous urine, free fat is dis-

charged in great quantity, either in the form of globules visible under the microscope, or more commonly, divided into molecules so small that they appear only as granular particles, under the highest magnifying powers. (See CHYLOUS URINE.)

3. The discharge of quantities of fluid fat by the kidneys is a phenomenon so extraordinary and unexpected, that its occurrence has been doubted. But there appear to be a few well authenticated instances. My colleague, Mr. Turner, informs me that such an instance fell under his own notice. The patient was taking cod-liver oil, and each day there was a discharge of yellow oil with the urine. Two examples have also been brought forward by Dr. C. Mettenheimer: one was a man with cancer of the lungs, who was taking daily a table-spoonful of cod-liver oil; the second was a woman convalescent from acute nephritis, who was taking the emulsio cannabina.[*] Dr. Henderson, likewise, describes three cases of heart disease, in which oil globules appeared on two or three occasions in the urine. (Brit. Med. Journ., 1858.)

4. Concretions containing fatty matter have been encountered in the urinary bladder. (See UROSTEALITH.)

5. *Kiesteine.* — This is a name given by Nauche to a peculiar pellicle said to form on the urine of pregnant women when left at rest for a few days, and said to contain fatty and caseous matter. Much has been written on the nature of this pellicle and its value as a sign of pregnancy; but the accounts are so contradictory that no safe conclusions can be drawn from them. I have carefully looked over all the observations hitherto made on the subject, and am inclined to believe that the kiesteine pellicle is nothing more nor less than the mould fungus which is apt to grow luxuriantly in urines containing organic matters. The urines of pregnant women are likely to form a fitting nidus for this fungus from the large quantity of epithelial débris which they generally contain. A very full account of the literature of this subject is given in

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[*] C. Mettenheimer—Archiv. d. Verein, Bd. i., p. 374. See also A. G. Long's Dorpat Thesis, *De adipe in urina et renibus*, &c. (1852). There is no doubt that cats and dogs, fed with an excessive quantity of fat, excrete oily matter in considerable proportion with the urine, so as to yield globules visible with the microscope.

Montgomery's "Signs and Symptoms of Pregnancy." A paper by R. C. Golding in the British Obstetric Record for 1847—48, and another by Hicks in the Lancet for 1859, vol. ii. p. 281, may also be consulted. The question deserves to be re-examined; but the investigation, to be of use, must be conducted with much more rigorous exactness than any hitherto published.

V.—PUS IN URINE.

Urine containing pus is turbid and milky when voided. After standing a while, it deposits a dense yellowish white sediment. Pus presents a very different appearance, according as the reaction of the urine is acid or alkaline. In the former case the deposit is loose, and the corpuscles discrete; but if the urine be alkaline, as it often is, from ammoniacal decomposition, the pus coheres into a viscid tenacious mass, which can be drawn out into long tough strings. This latter appearance is diagnostic of pus.

Micro-chemical characters.—Pus possesses an analogous constitution to blood, and is composed of cellular particles floating in a liquor puris. Liquor puris, like liquor sanguinis, is an albuminous saline fluid; therefore purulent urine necessarily contains more or less albumen—the quantity varying, according to the proportion of pus present, from a trace too slight for detection by ordinary reagents, to a considerable impregnation. It is sometimes a point of importance, and always of considerable nicety, to decide whether the quantity of albumen in a purulent urine is no more than can be accounted for by the pus present, or whether some of it is not due to renal disease. Such a question occasionally arises in cases of vesical calculi, accompanied with catarrh of the bladder—most surgeons holding that the co-existence of renal degeneration constitutes a bar to operation. Usually, purulent urines become merely hazy with nitric acid; and the quantity of pus must be very great indeed to account for a large deposit of albumen. Important assistance in doubtful cases is to be obtained by a diligent search for tube-casts in the freshly voided urine.

The chemical test for pus is the conversion of it into a viscid mass by the addition of liq. potassæ or liq. ammoniæ.

The pus corpuscle is a spherical cell, about one-third larger than a red blood-disk. Examined without reagents, it appears opaque, granular on the surface and yellowish (Fig. 24, *a*). The denser the urine the smaller and more crumpled becomes the pus corpuscle; whereas, the addition of water expands and clears it—sometimes bringing into view the nucleus. This effect is brought about much more quickly and powerfully by a drop of acetic acid, insinuated beneath the covering glass. The nucleus thus displayed is found to be cleft into two, three, and sometimes four nucleoli (*b*). If the acid be added in excess, the cell-wall and contents disappear altogether, and the cleft nuclei float free in the fluid.

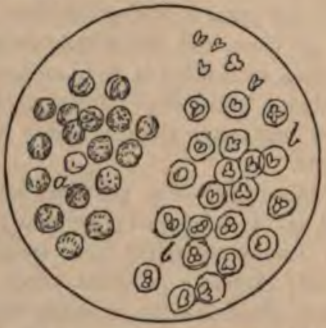

FIG. 24. Pus globules. *a*. Without reagents; *b b*. After the addition of acetic acid.

Clinical significance.—The importance of pus in urine depends on its source and quantity. Suppuration may take place in any part of the genito-urinary passages, or abscesses of adjacent parts may burst into these and cause pus to appear in the urine. It is therefore always desirable to decide the anatomical source of the pus. This is not always easy, and sometimes it is impossible. The following are the points to be held in view in such an inquiry:—

When pus is derived from the urethra (as in gonorrhœa) a drop or two may be squeezed from the meatus urinarius by compressing the penis. Gonorrhœa is the commonest cause of pus in the urine of men. The quantity is always small, and the general properties of the urine are not affected thereby.

In women the most common cause of slight admixtures of pus with the urine is leucorrhœa, which betrays itself by the co-existence of abundance of pavement epithelium.

Pus from the bladder has a more serious significance, as indicating the existence of cystitis. Usually there is little difficulty in tracing this to its right source by the local symptoms. In severe cases, the excessively frequent micturition—every ten or fifteen minutes—the ammoniacal state of the urine when voided, and the speedy gelatinisation of the pus into a viscid mass, leave no doubt on the mind of the practitioner. But when the cystitis is slight and of old standing, there is more difficulty, as the urine may retain its acidity, and micturition may not be very frequent. The presence of stone in the bladder, an enlarged prostate, the history of a past lithotomy, or of an old stricture, generally gives a key to the source of the pus.

Suppuration in the pelvis of the kidney (pyelitis) is generally indicated by direct signs of irritation in the loins. When these are absent, reliance must be placed on finding, with the pus, epithelial elements of transitional character (see Fig. 19), an acid reaction of the urine, and absence of signs pointing to the bladder and urethra.

The bursting of an abscess into the urinary passages is usually signalised by a sudden irruption of a large quantity of pus into the urine. Perineal abscesses opening into the urethra can scarcely be overlooked ; but perivesical and perirenal abscesses are more difficult to diagnosticate.

Purulent urine, from suppuration in the kidney, will come under consideration in future pages (See SUPPURATION IN THE KIDNEYS, PYONEPHROSIS).

VI.—BLOOD IN URINE—HÆMATURIA.

An admixture of blood with the urine is readily recognised by the colour which it imparts to the secretion, unless the quantity be very small. If the blood is derived from the kidneys it is diffused equally through the urine, communicating to it a reddish or a peculiar smoky tint, and after standing awhile a chocolate-coloured grumous deposit subsides. But when the blood is derived from some part of the urinary tract below the kidneys—ureters, bladder, or urethra—the colour imparted to the urine is pinkish or vermilion, and frequently distinct clots are found in the deposit.

The microscope is the surest means of discovering blood in urine; nevertheless the corpuscles may disappear very speedily if the urine be of very low specific gravity, or ammoniacal. In acid urine of moderate density (1020—25) the corpuscles remain visible and preserve their form for several days. They seldom present the bi-concave shape, but usually appear as simple circles (Fig. 25 *a*). Sometimes they shrink and crumple, and become misshapen in various ways (*b*).

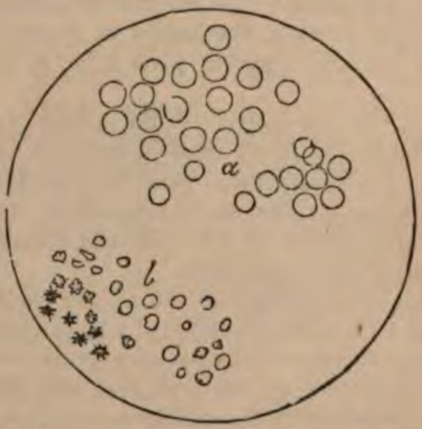

FIG. 25. Blood-corpuscles in urine. *a*. Slightly distended by imbibition. *b*. Serrated and shrivelled.

The marks by which blood-corpuscles are distinguished from other cells found in urine, are, the extreme tenuity of their outline, the absence of visible cell-contents, and especially of a nucleus, and their feeble refractive power. When the bi-concave form is preserved, this of course is diagnostic. Blood-disks are liable to be confounded with confervoid sporules, with the minute discoid forms of oxalate of lime, and with the nuclei of renal epithelium. From the first, they are distinguished by the absence of a nucleus, which can, with a good glass, always be detected in the sporules. Sporules also generally are somewhat oval, often elongated, and show signs of budding. The discoid crystals of oxalate of lime are distinguished by the existence of intermediate forms which connect them with dumb-bells. Renal nuclei are distinguished by their strong refraction, by being strongly tinted by magenta,* and usually they are surrounded by some portion of the material which originally invested them.

Urine containing blood is of necessity always more or less albuminous. The quantity may be so great that the urine looks like pure blood, and coagulates spontaneously, or so small that the microscope is required to detect it. The hæmorrhage may arise from a great variety of causes, which may be classified as follows :—

1. *Local lesions*—external injury, violent exercise, calculous concretions, ulcers, abscesses, cancer, tubercle, parasites, active or passive congestion, Bright's disease.
2. *Symptomatic*—in purpura, scurvy, eruptive and continued fevers, intermittent fever, cholera, etc., mental emotion.
3. *Supplementary or vicarious*—to menstruation, hæmorrhoids, asthma.

Cases also occur which are not referrible to any of these categories, of which the origin is extremely obscure.†

1. *Hæmaturia from local lesions.*—This division includes by far the largest number of cases. A point of great importance is to ascertain the exact source of the blood. This is not, as a rule, difficult.

Hæmorrhage from the *substance of the kidney* is recognised by the existence of tube-casts in the deposit. By far the most common cause of this variety of hæmaturia is some form of Bright's disease or its allies (congestion, &c.). In falls and blows on the loins, or any injuries supposed to implicate the kidneys, the occurrence of casts in the urine furnishes a valuable diagnostic sign. In the following remarkable case of laceration of the kidney from a fall, the condition of the urine was accurately noted from the time of the accident till death.

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* See a paper by the author, "On the Effects of Magenta and Taurin on the Blood-Corpuscles," in the Proceedings of the Royal Society for 1863.

† It may be necessary to remind students that in females the urine is generally bloody during the menstrual flow ; it may also become so at any time if there be uterine and vaginal hæmorrhage.

E. Davies, a bricklayer, aged 36, was brought into the Manchester Infirmary at 3 p.m. on April 27, 1863, in a state of complete insensibility, with gasping respiration, apparently dying. In the course of two hours he recovered consciousness, and answered questions imperfectly, in a half-drunken manner. It appeared that he went to his work in the afternoon intoxicated, and that he had fallen a height of seven stories. There was a compound fracture of the skull, and the legs were severely contused and lacerated. From the time that he recovered speech the patient continued to talk in a curiously incoherent manner, as if he were drunk—except that the pronunciation of words was unaffected.

No urine was passed on the day of the accident; but on the day following about eight ounces were withdrawn by catheter. The urine was excessively bloody, dark chocolate-coloured, and highly albuminous.

On the third day (April 29) the patient was in the same state. No urine was passed spontaneously; at 8 p.m. about an ounce was withdrawn by catheter; it was of the same character as before, but less bloody, and less albuminous. On the morning of the fourth day I found the patient breathing rapidly, with a quick small pulse; the tongue was moist; there was great thirst—no appetite; the bowels had been opened several times by medicine. At 9 p.m. of the same day I again visited the ward. No urine had been voided, and the bladder was not distended. The general condition was evidently worse; the delirium was constant, and he swore awfully when his legs were touched.

At noon on the fifth day the patient was much weaker; the breathing was interrupted; he muttered incoherencies unceasingly: and waved his hands as if he saw spectres in the air; he picked and tore the bed-clothes; he had torn three sheets to ribbons, and had torn the counterpane. He did this quietly, without violence, and without attempting to get out of bed. When asked questions he answered quite at random; the tongue was dry and red; pulse almost imperceptible. No urine had been passed spontaneously this day, nor the day before. The house-surgeon introduced a catheter, and succeeded, by compressing the abdomen, in withdrawing about two ounces of a yellowish urine, with small, dark, chocolate-coloured granules floating in it. About an hour after the patient died quietly, without coma or convulsions.

During the five days that the patient survived, no urine was passed spontaneously; but eleven ounces were withdrawn by catheter at three different times. The first specimen, drawn the day after the accident, was excessively bloody; the second, drawn on the third day, was much less bloody; the third, drawn just before death, contained no liquid blood, and had a yellow colour, but it deposited a considerable sediment of chocolate-coloured granules which consisted of indurated clots of blood. Although this last specimen, consisting of only two ounces, was the product of forty hours' secretion, its specific gravity was only 1015, and its proportion of albumen only $\frac{1}{12}$. The microscopic examination of the deposits revealed the existence of an immense quantity of casts of the uriniferous tubes, and these changed character as time passed over. In the first specimen the casts were all dark, opaque, and granular (Fig. 26, *a*, *c*, *e*,), evidently composed of crushed blood-clot; no free renal epithelium, nor any pyelitic cells, were found. In the second specimen, in addition to the dark granular casts, there were numerous deep-brown plain casts, with strongly-marked outlines and very few markings (*d*, *d*); a few transparent casts were also found, some of them studded with epithelium. In the second and third specimens free renal epithelium (*h*, *i*), and epithelium from the pelvis and infundibula (*g*), appeared in great abundance. The renal epithelium was deeply browned, evidently from hæmatine, but was otherwise natural. Many of the casts had dumb-bells imbedded in them.

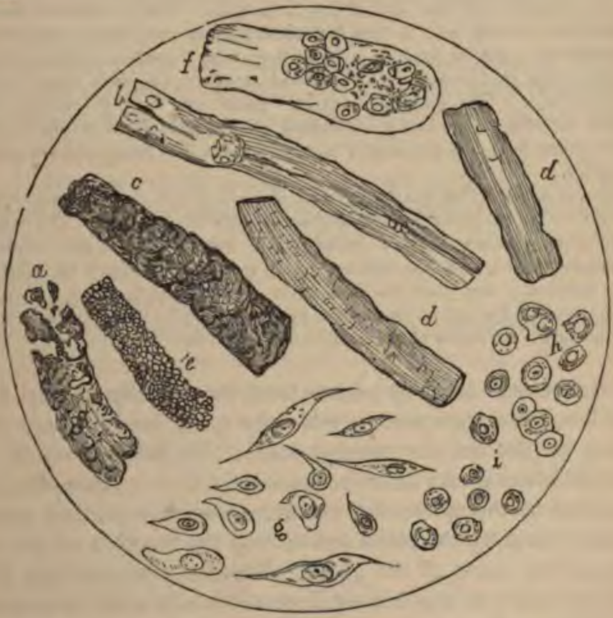

FIG. 26. *a*, *c*, *e*. Dark granular casts. *b*, *d*. Yellow plain casts. *f*. Large transparent cast studded with epithelium. *i*, *h*. Free renal epithelium, *i*, before, and *h*, after the addition of acetic acid. *g*. Cells from the pelvis and infundibula.

Autopsy forty-eight hours after death. Left parietal bone fractured, with a slight depression. Dura mater not lacerated; no free blood on or under the membrane; but there was an ecchymotic patch on it as large as a florin, corresponding to the fracture. There was no blood in the arachnoid space; but the pia mater was injected over the space of two square inches in the vicinity of the fracture. No lymph was thrown out on any part, nor was there softening or other abnormal condition of any portion of the brain.

Abdomen. There was no external sign of direct violence over the loins; all the abdominal organs, except the kidneys, were uninjured and healthy. *Left kidney* weighed 9¼ oz.; it was not lacerated. On section minute granules of indurated blood were found in several of the infundibula; the whole gland was hyperæmic. *Right kidney* weighed 9½ oz., was torn in two places on its posterior aspect. The lacerations ran across, somewhat crookedly, from the outer border almost to the hilum; they were about an inch apart, and varied in depth from one to three and even four lines. They were completely closed by a wedge-shaped solid clot of blood, which was very firm, and, where in contact with the renal substance, bleached. The renal tissue immediately adjacent to the lacerations appeared perfectly natural—neither injected nor softened. The tunica propria was of course torn through over the site of the lacerations. The lacerations did not penetrate in any part to the infundibula, but two large, firm, blood-concretions—one as large as a horse-bean, and the other as large as a pea—lay loose in the pelvis, and several smaller ones were found in the infundibula. The perirenal adipose tissue was deeply stained with blood on both sides; but it contained neither fluid blood nor clots. The peritoneum was not penetrated nor inflamed. The *heart* and *lungs* were healthy.

It was evident that the direct cause of death in this case was suppression of urine—aided perhaps by a degree of delirium tremens. The reason, probably, why no signs of inflammation were found in the brain and peritoneum was, that the patient never really rallied from the shock of the accident; and that reaction never properly took place. The desquamation of the epithelium of the pelvis and infundibula must be attributed to the irritation of the blood-concretion found therein.

Hæmaturia is rarely serious from its quantity in any form of Bright's disease, and is generally quite insignificant. Far more serious are the consequences of the coagulation of the effused blood in the uriniferous canals. Unless these plugs are expelled by the pressure of the urine from behind, they permanently block up the tubes and destroy the function of the corresponding parts of the gland. Hence any hæmorrhage from the substance of the kidney, however it may arise, is attended with serious hazard that the foundations of a fatal renal degeneration may be laid thereby.

Cancer of the kidney is often associated with profuse and repeated hæmaturia; the diagnosis rests chiefly on the presence of a tumour in the loins (see CANCER OF THE KIDNEY).

In tubercle, abscess, renal embolism, hydatids, the hæmorrhage is seldom more than trifling. In active hyperæmia of the kidneys after taking turpentine or cantharides, the bleeding is sometimes severe. As these classes of cases are treated separately in subsequent parts of this work, it will not be necessary here to go into further details.

Hæmorrhage from the *pelvis of the kidney and ureters* is commonly due to calculous concretions: much more rarely to cancer, tubercle, and parasites. When the blood has this source the diagnosis turns on the existence of symptoms of pyelitis, nephritic colic, and the passage of a foreign body down the ureter. Sometimes the blood coagulates in the ureter, and long vermicular clots may be afterwards recognised in the urine. The passage of these clots along the ureter produces precisely the same symptoms as a calculus passing in the same direction.

Hæmorrhage from the *bladder* is usually recognised by symptoms pointing directly to that organ, namely, excessively frequent micturition, pain in the hypogastrium, and at the neck of the bladder, &c. Exploration of the bladder will generally reveal the existence of calculi or fungoid growths. Varicose enlargement of the veins of the mucous membrane and acute cystitis are also occasional causes of vesical hæmorrhage.

Urethral hæmorrhage is known by the escape of blood in the intervals of micturition.

Symptomatic Hæmaturia. — Purpura hæmorrhagica is occasionally marked by severe hæmaturia. In a case under my care some years ago, there occurred first violent epistaxis requiring plugging of the nares; then profuse hæmaturia set in; when this subsided, the patient rapidly succumbed to intracranial hæmorrhage. Scurvy is more rarely attended with hæmaturia. The eruptive and continued fevers, cholera, and yellow fever, are sometimes the occasion of hæmaturia, which is generally a very unfavourable symptom.

Supplementary Hæmaturia.—Many curious examples have been recorded in which hæmaturia appeared to be *supplementary* to some natural function or some diseased condition. Chopart* relates a case in which hæmaturia supplemented a hæmorrhoidal flux; Latour† adds another. The latter mentions a singular case of spasmodic asthma, of such severity and persistence that the patient had not been able to lie in bed for eighteen months, which disappeared suddenly on the occurrence of hæmaturia. Chopart and P. Frank relate examples in which the menstrual flux was deviated to the urinary passages, and appeared under the form of a periodical hæmaturia.*

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* Traité des Malad. des voies urinaires. Segalas' edition, p. 283.

† Cited by Rayer, t. ii., p. 25.

The endemic hæmaturia of Mauritius, Brazils, Cape of Good Hope, Egypt, and some other hot countries, which has hitherto so greatly puzzled pathologists, seems at length to have found its explanation in the presence of a minute parasite which infests the mucous membrane of the pelvis of the kidney and the bladder. The researches of Griesinger, Bilharz, and Dr. John Harley on this subject will be described in the chapter devoted to parasites of the kidney (see BILHARZIA HÆMATOBIA).

Mental emotion seems capable in very rare instances of producing hæmaturia. Basham† tells of a shoemaker who was subject to attacks of hæmaturia which always recurred on the occasion of his drunken wife's misconduct. Rayer records an instance in which hæmaturia followed a fit of passion.

Treatment.—As hæmaturia is merely a symptom, and a symptom which attends a great variety of pathological conditions, the treatment of the cases in which it occurs is necessarily diverse. Sometimes, however, we are called on to treat hæmaturia for itself—in some cases because of our inability to fathom its exciting cause, in others because the loss of blood is so great that it becomes an urgent object to check it, even though the primary disease of which it is a symptom be irremovable.

In the hyperæmia of the kidneys which occurs in acute Bright's disease, after overdoses of turpentine and cantharides, after blows, falls, muscular efforts, and other external injuries, hæmaturia is a positive relief to the loaded vessels, and were it not that the effused blood is prone to coagulate in the uriniferous tubes, and produce a physical obstacle to the excretion of urine of a most dangerous character, the hæmorrhage (unless excessive) might safely be left to its own course. To relieve the congestion in these cases, derivation by the loins (cupping, etc.), by the cutaneous surface (baths, diaphoretics), and by the intestines (hydragogue cathartics), must be energetically practised.

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* Chopart (l. c. p. 282) cites one instance, and Rayer two instances, in which hæmaturia occurred at regular monthly periods in males. One of these was a butcher of Sedan. The circumstance became known, and such was the disgust caused thereby that no one would purchase meat from him.

† Basham on Dropsy, p. 259.

When hæmaturia is supplementary to hæmorrhoidal discharges, leeches may be applied about the anus. It should be remembered, however, that if the blood be shed from the mucous membrane of the bladder, and not from the substance of the kidney, such a discharge is not to be looked on unfavourably, nor to be rashly suppressed. When moderate hæmaturia occurs vicariously with menstruation, it is to be suppressed only on condition that the normal flux be re-established.

Passive hæmaturia *in the course* of zymotic diseases should be carefully distinguished from acute Bright's disease, which sometimes forms a *sequela* to these. In the former, the bleeding is probably from the whole or greater portion of the urinary tract, and not solely, if at all, from the kidneys. The internal remedies of most avail in passive hæmaturia, are the mineral acids, especially sulphuric acid, freely administered.

When our object is simply to treat the hæmaturia for itself —to stay the loss of blood—the first point is to enforce perfect rest, and to apply cold in the most effective manner to the bleeding part. If the kidneys be the source of the blood, ice-poultices should be applied to the loins; if the bladder, iced-water injections may be practised into the bladder, and iced-cloths applied to the perineum and epigastrium. The medicinal hæmostatics which have been found of most service, are gallic acid, acetate of lead, alum, ergot of rye, tincture of muriate of iron, turpentine, and matico. Dr. Golding Bird speaks highly of acetate of lead given frequently and in large doses for short periods. He recommends 3 or 4 grains, with one-fourth of a grain of opium, in a pill every two hours, until six or eight doses have been administered—care being taken to keep the bowels open by saline purgatives. Dr. Prout observes: "When the bladder becomes distended with blood, and complete retention of urine in consequence takes place, recourse must be had to a large-eyed catheter and an exhausting syringe, by the aid of which, and the occasional injection of cold water, the coagula may be broken up and removed. If the hæmorrhage be so profuse that the bladder becomes again distended with blood in a very short time, the injection of cold water into the rectum or bladder is sometimes of great use; and should these means fail, from 20 to 40 grains of alum may be dissolved in each pint of water injected into the bladder, a remedy that seldom fails to check the bleeding even when the cause is malignant disease. I have never known any unpleasant consequences follow the use of this expedient; and have seen it immediately arrest the most formidable hæmorrhage when all other means had failed, and when the bladder had repeatedly become again distended with blood almost immediately after its removal."*

Hæmatinuria (false hæmaturia).—Attention has been called by Vogel,† Oppolzer,‡ and Mettenheimer,§ to the escape of the colouring matter of the blood (hæmatine) with the urine, unaccompanied by rupture of the capillaries, and the presence of blood-corpuscles. The urine in such cases assumes a deep red or blackish red colour, very much as if it contained blood; but no blood-disks can be found under the microscope, nor any fibrin. This condition is invariably accompanied by the presence of albumen in the urine. It is caused by rapid destruction of the blood-disks in the bloodvessels, such as occurs in that state which is known as "a dissolved state of the blood," in septic, pyæmic and putrid fevers, and in some extreme cases of scurvy and purpura. In such cases hæmatine is set free by the disintegration of the red disks, and appears in the urine. Vogel found that inhalation of arseniuretted hydrogen produced an intense (but temporary) degree of hæmatinuria. He produced the same condition artificially in animals by inhalation of the same gas and of carbonic acid; also by the injection of substances into the veins which are known to dissolve and break up the red disks.

The clinical significance of hæmatinuria depends entirely on the pathological state which occasions it.

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* Prout: Stomach and Renal Diseases, 5th edit., p. 421.

† J. Vogel: Krankh. der Harnbereitenden organe, in Virchow's Handbuch der Speciellen Path. u. Therap., Band vi., 2te Abth., p. 539.

‡ Wiener Med. Wochensch. 1860, Nos. 25 and 26.

§ Würzburger Med. Zeitsch. 1862, p. 1.

VII.—CANCEROUS AND TUBERCULOUS MATTER IN URINE.

When cancer or tubercle of any part of the urinary tract has gone on to ulceration, the urine carries away with it some of the disintegrated elements, giving rise to an amorphous-looking grumous deposit. Sometimes masses of the morbid tissue as large as a horse-bean are discharged with the urine, and more or less blood is always mixed with such deposits.

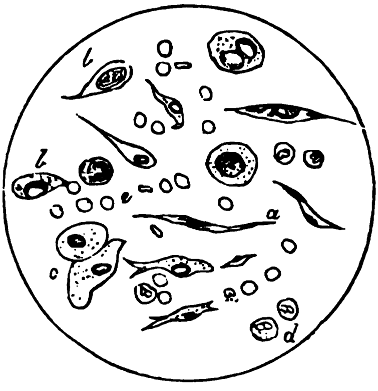

FIG. 27. Cells from the urine of a woman with fungus of the bladder. *a.* Fibro-plastic cells. *b, b.* Cancer cells. *c.* Epithelial cells. *d.* Pus. *e.* Blood.

Very great caution is requisite in coming to a conclusion as to the *cancerous* nature of cells found in urine, on account of the great similarity between the irregular transitional forms of the epithelial cells lining the urinary passages, and the cells of cancerous growths. Indeed it would be quite unsafe, in such a case, to rely on the mere form and size of individual cells. In the above drawing may be seen the diverse shapes discharged with the urine in a case of malignant fungus of the bladder. If the forms be compared with those in Figs. 19 and 26*g*, the similarity of the cells will appear very striking. It is more safe to take the entire character of the deposit into consideration. It may be described as a thick, dirty, blood-stained sediment, containing abundance of blood-corpuscles, mixed with spindle-shaped, oval, and irregular cells. Pus-corpuscles are either wholly or nearly absent. The presence of shreds or pieces of solid tissue appreciable to the naked eye, should be carefully looked-for: their occurrence is almost a certain proof of the existence of some morbid growth. The character of the deposit generally, and especially the presence of numerous spindle-shaped (fibro-plastic) cells, which cannot be mistaken for epithelial elements, indicate clearly that some morbid growth or natural tissue is being broken up. The collateral symptoms are then generally sufficient to decide whether the broken-up tissue is a portion of the natural membranes or an adventitious growth. In cancer of the kidney no help to the diagnosis must be expected from the character of the urinary deposit (see CANCER OF KIDNEY).

The discharge associated with *tuberculous* ulceration differs from that of a cancerous fungus in being largely purulent; indeed, pus-corpuscles are often the only appreciable formed elements in the urine in cases of tubercle of the kidney and bladder. But in other cases, broken-down cheesy masses may be seen, together with a large quantity of amorphous, or barely morphous granular débris (see TUBERCLE OF THE KIDNEY).

It follows, of course, that cancerous and tuberculous masses may exist in the kidney, or beneath the mucous membrane of the urinary passages, without contributing anything to the stream of urine. It is only when ulcerated that their elements escape with the urine; before this takes place they may, however, give rise to copious and oft-repeated hæmorrhage.

VIII.—SPERMATOZOA IN URINE—SPERMATORRHŒA.

The admixture of semen with the urine gives rise to a mucous-looking deposit. When in large quantity, white albuminous flakes and masses are seen; these exhibit a viscid consistence when taken up with the pipette. The microscope reveals the existence of spermatic filaments, consisting (Fig. 28) of a minute oval head, not more than $\frac{1}{10.000}$ of an inch in breadth, and a long whip-like tail of extreme delicacy. The length of the entire filament is $\frac{1}{600}$ of an inch.

When freshly shed, and still living, they exhibit active eellike movements, strongly suggestive of volition ;* but as seen in urine they are always motionless. They offer considerable resistance to disintegration, and may sometimes be recognised in decomposed urine which has been kept for weeks.

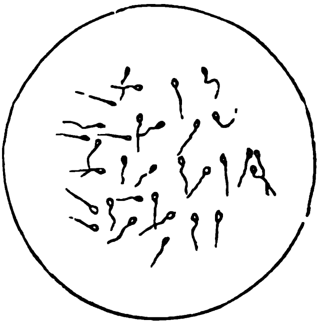

FIG. 28. Spermatozoa.

A certain quantity of seminal fluid necessarily finds its way into the urine of both sexes after coitus; also into the urine of men after involuntary nocturnal emissions.

Involuntary nocturnal emissions occurring occasionally in the young and continent, are not to be regarded as within the limits of disease ; but when they take place two or three times weekly or oftener, or when the acts of defæcation and micturition are frequently followed by a glairy discharge, a diseased state must be acknowledged to exist; and one also, as experience proves, exceedingly difficult to deal with. Whether it be that the mental phenomena observed in these cases are altogether secondary to the genital defect may well be questioned ; but it is an important—indeed *the* important—fact in relation to involuntary seminal discharges, that they are associated with a deplorable state of mind. Much of this is no doubt owing to the prurient eagerness with which persons so afflicted seek satisfaction to a fatal curiosity, in the publications of unprincipled quacks, who lure their victims with libidinous descriptions, and afterwards terrify them with exaggerated and lying pictures of the fate which awaits them.

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* Students may be reminded that spermatozoa are not really independent animals, but simply the escaped contents of a cell. They are floating cilia, and resemble the oscillating sperm-cells of the antheridiæ of mosses.

But there is a danger that the legitimate practitioner may come to look upon cases of this class too lightly, and thus be the indirect occasion of their seeking the help which is their injury.

The least serious cases are those in which the emissions are solely nocturnal. As long as the complaint is confined within these limits the general health does not suffer, and the mental state is seldom gravely disturbed. Sometimes, however, individuals of fervid imagination, whose health is from any cause below par, fix upon this incident (nocturnal emissions) with fatal tenacity, and hinge their ill-health entirely upon it, when in reality it has nothing to do with the matter. Persons go on for years subject to nocturnal pollutions without consciousness of any harm resulting, but when they chance to become dyspeptic, or their nervous system becomes upset by overwork, *then* these emissions loom largely to their imaginations, and they connect them with their failing health.

When seminal discharges occur daily, and accompany or follow defæcation and micturition, a greater departure from the natural state is betrayed; and it is seldom that such a state of things continues for any length of time without inducing pallor, weakness, want of zest and energy for work, as well as a fidgetty, vacillating, and sometimes very depressed state of mind. Nevertheless, these consequences frequently altogether fail. There was recently a patient under my care at the Royal Infirmary—a ruddy strong-looking young man of six-and-twenty—who had been in the habit, according to his own account, for the last seven years, of discharging large quantities of seminal fluid almost daily, more especially with micturition. In a specimen of his urine brought to me, there was at least a table-spoonful of glairy matter having the microscopic and other characters of semen. The mental state was certainly shaken, but solely, as it appeared to me, from the diligent study of Mr. Dawson's book on spermatorrhœa. He talked with a sort of gloomy satisfaction of being tired of life, but it was with an air as if he were repeating a lesson, and not as one revealing a terrible conviction.

The type of mental disturbance usually associated with spermatorrhœa, is common in this as in other large towns, independently of seminal losses among persons—chiefly men of business—whose health has given way from too engrossing application to exciting pursuits. Such persons became nervous, apprehensive about themselves to a distressing degree, pusillanimous, subject to attacks of incomplete syncope; they lose their sleep, and sometimes their appetite; there is some real emaciation and a great deal of fancied wasting. They pour into the ears of their medical attendants an endless variety of symptoms, and worry them beyond the most tedious hysterical women. Such patients, although often men of middle age, or at least beyond their first youth, and fathers of families, rarely fail to complete the catalogue of their ailments with a reference to what they conceive to be some anomaly of their sexual functions.

Involuntary discharges are not confined to youth or middle age. Men advanced in years are sometimes tormented in the same way, and exactly the same state of mind is observed in them. They imagine their "substance" to be ebbing from them, and their virility departing. A gentleman over sixty years of age, the father of a family of married daughters, was so concerned about a slight seminal discharge which in no way affected his health, that he forwarded to me for examination over a hundred specimens of his urine.

In the *treatment* of this class of cases, the first point to establish is whether the trouble of the nervous system is the primary phenomenon, and the disturbance of the sexual functions only an insignificant incident, or whether the seminal losses are in such frequency and quantity that they may be regarded as having a hand in evolving the symptoms complained of. The great majority of cases belong to the former category; and indications for treatment are to be looked for in the general state of the patient and the circumstances surrounding him, rather than in the condition of the sexual functions. If it appear, after a patient sifting of the actual phenomena and the past history of the case, that the seminal emissions must be regarded as the fundamental ailment, the next point is to inquire into the existence of any local cause for the emissions.

The irritation of ascarides or hæmorrhoids sometimes occasion involuntary discharges: also herpetic eruptions about the prepuce. Lallemand enumerates a long prepuce as contributing to the same, by the lodgment which it affords, in uncleanly persons, to offensive secretions. Whatever be the local cause discovered, its immediate removal is of course the first step in the treatment.

In the absence of a local cause, the evil can usually be traced to venereal excesses, masturbation, and the reading of salacious literature. Some of these cases are very difficult to deal with. An attempt must first be made to put a stop to the practice which is the cause of the complaint. The further treatment should be directed to improving the tone of the muscular system by daily ablutions with cold water or brine, by sea bathing, regulated exercise, change of air, &c. The state of the patient's mind often requires that the time, quantity and material of the meals shall be minutely regulated. The diet should be nourishing and bland: spices and condiments should be avoided. Malt liquors and the lighter wines are to be cautiously employed; the quantity must be judged by their effects. Any quantity which produces flushing of the face is too much. An opiate sometimes renders good service by securing a good night's rest. Astringent and ferruginous tonics offer valuable aid to the hygienic treatment. Tincture of the muriate of iron has appeared to me to produce a better effect than any other preparation. A blister to the perineum has sometimes seemed to diminish the emissions. In cases of inveterate masturbation, Mr. Helton found that he could invariably put a stop to the practice by applying a strong solution of iodine or blistering fluid to the penis so as to render the organ too sore for manipulation.*

Lallemand recommends the local application of nitrate of silver to the orifices of the ducts of the vesiculæ seminales by means of his porte-caustique. I cannot say that I have ever seen cases in which this severe proceeding seemed justifiable. It must be remembered that it is not without danger. Dr. Bird relates an instance in which a dangerous cystitis was produced in a healthy person by the local application of the solid nitrate of silver in this manner. Dr. Chambers has communicated another and more untoward example, in which death followed the application of an irritant ointment by means of a catheter in a case of imaginary spermatorrhœa.[*]

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* Lancet, 1863, II. 123.

Dicenta and B. Schulz speak in high terms of the constant galvanic current. Schulz directs the current to be transmitted along the vertebral column for one or two minutes, and repeated three or four times a week. Twenty or thirty Daniel's elements, of medium size, should be used; the positive pole should be applied to about the fifth dorsal vertebra, and the negative to the sacrum or perineum.[†]

IX.—CONFERVOID VEGETATIONS IN URINE.

Minute vegetations are apt to make their appearance in urine, and to cause confusion in its examination. It is therefore necessary that the student should be familiar with their appearances, so that he may not mistake them for objects of more importance. With one exception (sarcinæ) they are not present in urine when voided, and their existence is a sign and accompaniment of those changes on keeping which eventually lead to putrefaction. The following deserve a separate notice, namely, *vibriones*, the *mould fungus*, the *sugar fungus*, and *sarcinæ*.

1. *Vibriones.*—These are the simplest in structure and the most common. They consist of minute linear particles about $\frac{1}{3000}$ of an inch long, incessantly moving, which swarm in infinite myriads in urine that is beginning to putrefy (Fig. 29). Their appearance is certain evidence that putrefactive changes have set-in; the urine loses its transparency and deposits a cloudy sediment; its odour becomes offensive, and its reaction soon ammoniacal. By a very high magnifying power these moving filaments are resolved into a series of granular particles arranged in line.

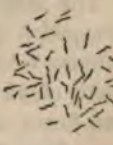

FIG. 29.
Vibriones in urine.

[*] Lancet, 1861, p. 582.

[†] Year Book, 1863; p. 300. Report on Surgery.

2. *Mould fungus* or *Penicilium glaucum.*—Like other organic fluids, urine is liable to mildew. Dr. Hassall has shown that urine may mould from the growth of two distinct, though closely allied, vegetations. The first of these is the penicilium glaucum, or common mould which grows in vinegar and all albuminous fluids; and the second is the yeast or sugar fungus which flourishes in diabetic urine.

The mould fungus may be found in urine in the three phases of its development, namely, as round and oval cells or *sporules;* as an interlacement of fibres, or *thallus;* and as a downy pile of threads growing into the air, or *aerial fructification.* This last phase is, however, not seen unless the urine has been kept several days.

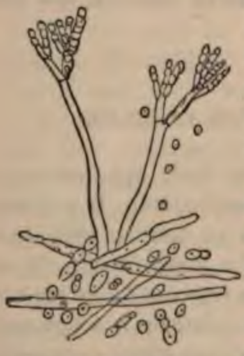
Fig. 30. Mould fungus.

The sporules often appear in urine a few hours after emission. It is important to be familiar with their microscopic appearance to avoid confounding them with blood disks. The marks by which they are distinguished are: the great difference of size among the individual cells; the presence of a nucleus in the larger sporules; their tendency to assume an elongated or oval form; and the indications of budding and commencing formation of a thallus.

The interlacing fibres of the thallus are produced by the elongation and gemmation of the sporules, and are composed of tubular cells placed end to end. This interlacement forms a fleecy cloud in the urine, which gradually rises to the surface and forms an islet or patch of mould from which spring the ascending stems of the aerial fructification. The latter consist of hollow filaments rising from the thallus, which divide at their extremities into two or three branches; these again subdivide into a number of digitate projections so as to form an irregular tuft or head (Fig. 30). The digitate projections are filled with sporules, and eventually burst, giving exit to the sporules, which then fall into the urine below, and collect into an amorphous-looking deposit at the bottom of the vessel.

The growth of this vegetation in urine has been admirably described by Hassall (Med. Chir. Trans., vol. xxxvi. p. 32) with the conditions which favour and impede its development. The urine must be acid; when it becomes ammoniacal, the further growth of the plant is arrested, and it soon perishes. The presence of some organic matter is another necessity; but as the urine is never absolutely free from organic matters (albumen, epithelial scales, pus, &c.), it may be said that every acid urine forms a fitting nidus for the mould fungus. Albuminous acid urines are those in which the plant grows most luxuriantly.

3. *Yeast or sugar fungus* (Torula cerevisiæ).—This vegetation has precisely the same phases of development as the mould fungus; and in the phases of sporule and thallus, it is not easy to distinguish the one from the other; but the aerial fructifications of the two are wholly different. The yeast fungus (which grows luxuriantly in diabetic urine exposed to the air at a moderate temperature), instead of a tuft of branches has a spherical head (Fig. 31). When the plant has attained its full fructification, the floating bed of thallus appears dusted over with a brown powder. Under the microscope the brown matter is found to consist of the spherical heads full of sporules. These, when ripe, burst, and discharge their sporules, which sink to the bottom of the glass and form a white settling like so much flour.

Dr. Hassall believes that the growth of the sugar fungus in urine is a certain proof of the existence of sugar. Whether it be so or not, is scarcely capable of absolute proof, until it shall have been shown that the natural urine is wholly free from traces of sugar. The yeast fungus may grow even to full fructification, as I have repeatedly witnessed, in urine in which the most delicate direct testing has failed to detect sugar.

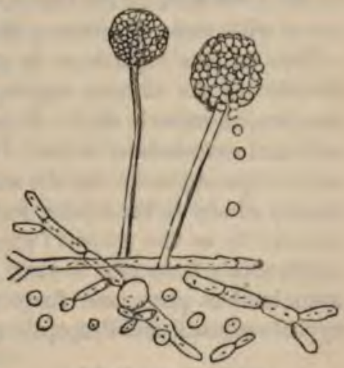

FIG. 31. Sugar fungus.

4. *Sarcinæ* (Fig. 32).—Since Heller and Mackay, in 1848, first discovered sarcinæ in urine, they have been observed by Johnson, Beale, Welcker, Munk and Begbie. The seat of production of this vegetation is probably the bladder; and it is discharged with the urine, sometimes in great quantities, and forms a greyish-white amorphous-looking deposit. It consists of the same elements as the sarcina ventriculi (of Goodsir), and is usually regarded as the same species. Both the cubical masses and their component particles are, however, smaller than those of the gastric sarcina, and Rossmann and Welcker[*] consider these differences sufficient to establish a specific distinction. It seems more probable, however, that the differences in the habitat and conditions of growth are sufficient to account for the diversity of size. Dr. P. Munk[†] has shown that one of the points relied on by Welcker, namely, the absence (in urinary sarcina) of cubes containing more than 64 particles, is not constant. Munk found cubes of 512 particles. In some vomited matter sent to me for examination by Dr. Scowcroft, of Southport, I detected small-sized sarcinæ mixed with those of ordinary dimensions.

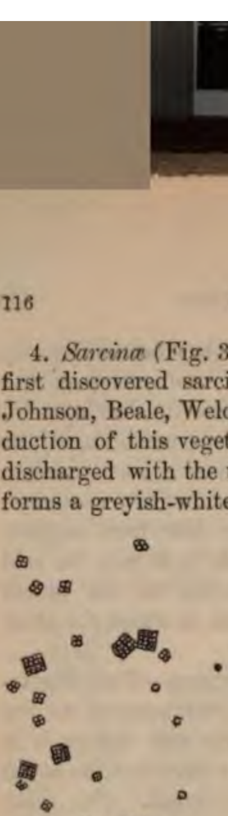

FIG. 32.[‡]
Sarcinæ in urine.

This curious vegetation is generally associated with some disorder of the urinary organs (renal pains, painful micturition, vesical catarrh, &c.). It grows, or at least exists, both in acid and ammoniacal urine. In Munk's case the fungus grew in great quantities during the summer months, and disappeared almost wholly in the winter months; and this was the more remarkable as the patient (who was paraplegic) kept his bed continuously from year to year. Dr. Begbie's patient suffered from lumbar pains and frequent micturition, together with hypochondriacal and dyspeptic symptoms.[§]

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[*] Ueber Sarcina im Urine des Menschen. Henle and Pfeuf. Zeitsch. 3tte. R. Bd. V. 199.

[†] Ueber Harnsarcine—Archiv. f. Path. Anat. 1861, p. 570.

[‡] After Welcker, Henle and Pfeufer's Zeitsch. Bd. V. taf. x.

[§] Edin. Med. Journ. 1856—7.

No treatment yet tried has had any appreciable effect in checking the growth of sarcinæ in urine.

X.—ALBUMEN IN THE URINE.

Albumen is not found in any proportion in healthy urine; but it is the most common and the most important of the abnormal ingredients found in disease. Its presence in the urine is due to so many causes that the fact itself yields little direct information; but when correctly interpreted, it furnishes a key to several grave pathological states which would otherwise remain in great obscurity.

Tests for albumen.—The tests for albumen are coagulation by heat or nitric acid; in doubtful cases the two should be used together.

Heat.—If the urine have its usual acid reaction, it becomes turbid on boiling when it contains albumen; and this turbidity persists after the addition of an acid. The best way of operating is to fill a test-tube to the depth of about an inch with the suspected urine, and to apply the flame of the spirit-lamp to the upper stratum of fluid until it boils. The lower part preserves its original transparency, and thus serves, by contrast, to indicate more clearly any change that has taken place in the heated portion. When the urine is cloudy from amorphous urates, heat *alone* is a complete—and the best—test for albumen. The precipitation of the urates is sufficient evidence of an acid reaction: when the urine is warmed the urates are speedily dissolved and the urine becomes transparent, but as the temperature approaches the boiling point it becomes again turbid if it contain albumen.

Highly albuminous urines begin to coagulate at a much lower temperature than feebly albuminous ones, and when the quantity of albumen is only a trace, turbidity does not occur until the urine has begun to boil.

There are two points to be remembered in using heat alone as a test for albumen. First, that albumen is not coagulated by heat when the urine is alkaline; in such cases, therefore, it is necessary, before boiling, to restore the acidity by a few drops of acetic acid. Secondly, when the urine is neutral, or very feebly acid, it may become turbid on heating from precipitation of the earthy phosphates; but turbidity from this cause is easily distinguished from albumen by a drop of acetic or nitric acid which instantly causes the phosphates to disappear.

Nitric acid.—Nitric acid is an extremely delicate test for albumen. The best manner of applying it is to fill a test-tube to the depth of about an inch; then, inclining the tube, to pour in strong nitric acid in such a manner that it may trickle down along the sides of the tube to the bottom, and form a stratum some quarter of an inch thick below the urine. Added in this manner there is scarcely any mingling of the two fluids, and if albumen be present, three strata or layers will be observed: one, perfectly colourless, of nitric acid at the bottom; immediately above this an opalescent zone of coagulated albumen; and atop the unaltered urine. If there be only a trace of albumen, two or three minutes elapse before the opalescent zone becomes visible.

There is no method equal to this for detecting minute quantities of albumen. The reaction of the urine does not interfere with its operation. Only one caution is necessary. In concentrated urines, and especially febrile urines, the addition of the acid is apt to precipitate the amorphous urates, and thus to occasion a turbidity which might be mistaken for albumen. The two conditions are however easily distinguished by observing the level at which the cloudiness begins, and the direction in which it spreads. Albumen begins to coagulate immediately above the stratum of acid, and the turbidity spreads upwards; but the urates first appear at or near the surface of the urine, and the opacity spreads downwards. Heat also readily resolves the doubt; for the urates speedily disappear when the urine is warmed, but turbidity from albumen is not affected by heat.

The urine of patients who are taking cubebs and copaiba is commonly somewhat opalescent, and nitric acid, in the cold, sometimes (not always) increases the opalescence. The sense of smell immediately directs attention to the presence of these drugs, and heat diminishes the opalesence and prevents any turbidity with nitric acid.

In urines which are over rich in urea, nitric acid, in the cold, causes a slow precipitation of a crystalline mass of nitrate of urea, which however is so different in appearance from coagulated albumen that it can scarcely be mistaken for it.

It is further to be remarked, that if the manner above described of testing for albumen with nitric acid be not followed, two notable fallacies may be encountered. On the one hand (as was pointed out by Bence Jones), if the urine be acidified with a small quantity, a drop or so, of nitric acid, the albumen may not be coagulated at all ; and on the other hand, if a large quantity of acid (an equal volume) be suddenly added to, and mixed with the urine, the mixture remains perfectly clear, even though it may be highly albuminous. I have known the latter fallacy occasion concealment of albumen in the urine, in a case of Bright's disease, for many months.

In practice, the best method of proceeding is as follows: when the urine is cloudy from urates, use heat alone ; when the urine is clear (or in any case if alkaline) add nitric acid in the way above described ; if no turbidity appear above the layer of acid, the urine may be with certainty pronounced free from albumen ; if a turbid zone is produced, apply heat thereto ; if it be not dissipated, albumen is certainly present.

Several other substances (bichloride of mercury, sulphate of copper, alum, ferrocyanide of potassium, creasote, carbolic acid and alcohol) coagulate albumen ; but they are unsuitable for urine-testing, because those of them which are miscible with it, precipitate also some of the natural constituents of the urine.

The *quantitative* estimation of albumen in urine is a matter of considerable practical importance, and it is to be regretted that no exact and easy method of attaining this object has been devised.

For precise determinations the plan usually followed is to bring a measured quantity of urine to a slightly acid condition ; boil ; throw on a weighed filter ; wash ; dry at 212° ; and weigh. This proceeding demands a good deal of time. The filtering is sometimes impossible ; and the results obtained are only moderately accurate with every care.

For a rough and ready, but very useful, method, there is none superior to boiling the urine in a test-tube with a drop or two of acetic acid. The albumen coagulates in flakes, and presently sinks to the bottom, forming a layer of various thickness. The proportion of albumen is judged of by the depth of this layer as compared to the height of the column of urine in the tube. This proportion may be expressed in numbers, as $\frac{1}{2}$, $\frac{1}{4}$, $\frac{1}{12}$ and so forth. If the quantity of albumen be too small to form a layer of appreciable depth, the proportion is expressed more loosely, as a "cloudiness" or an "opalescence." The varying density of albuminous urines, and the varying size of the flakes into which albumen coagulates, affect the rapidity and completeness of the subsidence, and therefore the depth of the coagulated layer, so that only approximate results can be expected from this method.

Becquerel ingeniously turned to account the property of albumen to deviate the plane of polarization to the left: and constructed an instrument on a similar plan to the optical saccharimeter, by which the deviation could be measured, and the per-centage of albumen calculated therefrom. It would appear, however, that this instrument, on Becquerel's own showing, is only capable of very limited clinical application. When the quantity of albumen is considerable it gives very exact indications; but the deviation is too slight for exact estimation in moderately and feebly albuminous urines; it is therefore useless for the bulk of albuminous urines.*

Boedecker has recently proposed a volumetrical method, founded on the property of ferrocyanide of potassium to form an insoluble compound of fixed composition with albumen. Vogel states that he has found this method inaccurate.†

Modified albumen in urine.—Dr. Bence Jones has described a modification of albumen, which he designated hydrated deutoxide of albumen. He found it in the urine of a patient suffering from mollities ossium. The urine did not give a precipitate with nitric acid alone, nor by boiling, nor by adding nitric acid to the boiling urine. But if the urine was boiled, and then allowed to cool, a precipitate fell; and this was immediately redissolved by heat. The same substance has been found in the buffy coat of inflamed blood, and it may also be met with in the albuminous fluid of pus (Bence Jones, Animal Chem. p. 109).

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* See a clinical lecture by Becquerel—Clinique Européenne, 1859, p. 54.

† Boedecker's method is described in Henle and Pfeufer's Zeitsch. 1859, p. 321.

Clinical significance of albumen in the urine.—In considering this subject all those cases are, of course, excluded in which the occurrence of albumen is only incidental to the presence of some other fluid in the urine, such as blood, pus, or spermatic fluid.

The excessive use of a diet composed exclusively or chiefly of albuminous matters, such as eggs, has been shown by Barreswil, Brown-Sequard and others to cause the urine to become slightly albuminous. Bernard found that irritation of the renal nerves and of a certain spot in the floor of the fourth ventricle (higher up than the diabetic puncture) caused albumen to appear in the urine of animals. The same occurred when albumen of eggs was injected into the veins, and even when a large quantity of pure water was so injected (Magendie Kierulf). Interference with the circulation of the blood in the kidneys produces albuminuria (see Congestion of the Kidneys). Vogel found that inhalation of arseniuretted hydrogen and carbonic acid caused the urine to be abundantly albuminous.*

Slight and temporary albuminuria appears to occur, in highly exceptional cases, from very slight disorders. Beneke, when suffering from dyspepsia, noticed albumen in his own urine four times in as many weeks. Similar observations have been made by others (Parkes).

Setting aside these unimportant exceptions, albuminuria must always be looked on as a grave symptom of disease; and when discovered, it becomes an anxious question to the practitioner: What signification has it?

The pathological states in which albumen appears constantly or occasionally in the urine may be arranged into the following groups :—

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* Krankh. d. Harnbereitenden Organe, p. 518. It has also been asserted that the urine becomes albuminous in mercurial salivation. This assertion was long since shown to be erroneous by Rayer. More recently Dr. Francis, under the supervision of Dr. O. Rees, examined the urine of fifteen salivated individuals without finding a trace of albumen.—(On "Diseases of the Kidneys," by G. O. Rees, p. 28. London, 1850.)

1. Acute and chronic Bright's disease of the kidneys.
2. Pregnancy and the puerperal state.
3. Febrile and inflammatory diseases (zymotic diseases, such as scarlet fever, measles, small pox, typhoid, cholera, yellow fever, ague, diphtheria, etc.; inflammatory diseases, such as pneumonia, peritonitis, traumatic fever, articular rheumatism, etc.)
4. Impediments to the circulation of the blood (emphysema, heart disease, abdominal tumours, cirrhosis, etc.)
5. A hydræmic and dissolved state of the blood and atony of the tissues (purpura, scurvy, pyæmia, hospital gangrene).
6. Saturnine intoxication.

In the first group albuminuria is dependent on structural changes in the kidneys (see BRIGHT'S DISEASE).

In the second group albuminuria is sometimes associated with structural changes, and sometimes not (see CONNECTION OF BRIGHT'S DISEASE AND PREGNANCY).

In all febrile and inflammatory complaints a trace of albumen is occasionally found in the urine; it usually amounts to no more than a trace, and disappears on defervescence; sometimes in pneumonia it is not inconsiderable. As intercurrent febrile attacks are common in the course of most chronic complaints, temporary albuminuria has been noted in a great multitude of different diseases. This remark applies especially to chronic tuberculosis, cancer, caries and necrosis; and albuminuria under such a condition is to be carefully distinguished from the cases in which genuine Bright's disease coexists with those complaints. In a zymotic disease there is a double pathological state, namely, pyrexia and the operation of a specific poison; and albumen may appear in the urine either as an incident of the febrile state, when it is comparatively unimportant, or as an indication of serious structural changes in the kidneys, which constitute a grave sequela of the disease.

Albuminuria connected with impediments to the circulation of the blood is considered under CONGESTION OF THE KIDNEY.

In a dissolved or putrid state of the blood, albumen appears in the urine without being connected with organic changes in the kidney; it is associated with the escape of the colouring matter of the blood (see HÆMATINURIA).

The occurrence of albumen in the urine of persons poisoned with lead, although repeatedly observed, was not regarded as anything more than a coincidence until Ollivier demonstrated, by experiments on animals and clinical observations, the existence of a causal connection between them. Ollivier[*] found that dogs, rabbits, and guinea pigs when poisoned with repeated doses of carbonate of lead, invariably passed an albuminous urine, and that their kidneys exhibited signs of incipient organic disease. He also collected 15 examples of albuminuria in persons poisoned with lead. Seven of these had temporary albuminuria; in three, the albuminuria persisted during the continuance of the saturnine symptoms; and in four, genuine Bright's disease had been produced. In addition to these observations, he examined the urine of 37 persons affected with diverse manifestations of lead poisoning in the Hôpital de la Charité: of these, 9 had albuminous urine. These observations have been confirmed by Lancereaux[†] and Danjoy.[‡] Ollivier found that both the urine and the kidneys in these cases contained traces of lead. He inferred that the existence of lead in the kidneys induced an organic lesion of these organs, and that the albuminuria was the consequence of that lesion.

When albumen is found in urine, the important point to decide is, whether it indicates the existence of organic disease of the kidneys or not. This question, in any individual case, must be considered chiefly in connection with the three following points jointly, namely:—

1. The temporary or persistent duration of the albuminuria.

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[*] *Archives Générales*, 1863, II. pp. 530 and 709.

[†] *Union Médicale*, 1863, and *Bulletins de la Société médicale d'Émulation* —nouvelle série, t. 1, p. 182, 1864.

[‡] *Archives Générales*, 1864, I. p. 402.

2. The quantity of the albumen; and the occurrence and character of a deposit of renal derivatives.
3. The presence or absence of any disease outside the kidneys which will account for the albuminuria.

1. Dr. Parkes* has collected some instructive facts on the importance of distinguishing between temporary and permanent albuminuria. He gives a tabular statement of the adult cases treated by him in University College Hospital, in which the urine was examined carefully and daily for a sufficient length of time, to enable it to be said with perfect certainty that albumen was or was not present during the whole course of the disease, or during any part of it. Cases of cystitis and vaginitis were not included. By the term "temporary albuminuria," Dr. Parkes implied cases in which albuminuria, after lasting some days or even weeks, disappeared entirely for some time before the patient left the hospital; and by "permanent albuminuria," cases in which the albumen did not disappear during the time the patient was under observation—this time being generally very long, and always many days. The cases were of the miscellaneous character usually admitted into a London hospital. Cases of cholera and pregnancy were excluded. The total number of cases tabulated was 303—170 men and 133 women. The results are given separately for the two sexes by Dr. Parkes; but as no difference of any moment to the inquiry were found between the sexes, I have taken the liberty of throwing the numbers together, for the sake of simplicity, in the following tables:—

TABLE I.

Total number of cases.	Urine not albuminous at any time.	Urine temporarily albuminous.	Urine permanently albuminous.
303	227	37	39

Reduced to per-centages:—in 75 the urine was never albuminous; in 12 temporarily albuminous; and in 13 permanently albuminous.

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* On the Composition of the Urine, p. 186.

The 37 cases of temporary albuminuria occurred in the following diseases:—

TABLE II.

Disease.	Total number of cases.	Temporary albuminuria.	No albuminuria.
Paraplegia (spinal) . .	2	1	1
Hemiplegia (cerebral) . .	4	2	2
Chronic phthisis . .	28	1	27
Pleurisy (acute) . . .	17	1	16
Bronchitis (acute, simple) .	6	1	5
Bronchitis (acute in emphysematous lungs). . .	9	2	7
Bronchitis (in tuberculous lungs)	1	1	—
Pneumonia (acute, lobar) .	10	6	4
Heart disease (organic) . .	17	2	15
Morbus Brightii (acute) .	3	3	—
Acute rheumatism . .	19	4	15
Subacute rheumatism .	8	1	7
Purpura hæmorrhagica . .	1	1	—
Typhoid fever . . .	19	5	14
Typhus	2	1	1
Variola	5	2	3
Scarlatina	2	2	—
Erysipelas (leg) . . .	1	1	—
Total	154	37	117

The quantity of albumen was "large" in the cases of pneumonia and acute Bright's disease; was in "some quantity" in the cases of typhoid, variola and scarlatina, and "very small" in most of the remainder.

The diseases in which permanent albuminuria was found are detailed in Table III. on the next page. According to this table, in 32 out of 39 cases of permanent albuminuria, disease of the kidney was either proved to exist or rendered highly probable by other symptoms; and as 3 other cases are excluded because there was no decided information as to the state of the kidneys, permanent albuminuria indicated renal disease in 32 cases out of 36; and if heart diseases are excluded, it indicated renal disease invariably.

TABLE III.

Disease.	Number of cases.	Remarks.
Morbus Brightii (all forms) .	25	
Encephaloid disease of kidney .	1	
Cystic disease of kidney . .	1	
Leucocythemia, with presumed lardaceous kidney . .	1	
Chronic phthisis and kidney disease (autopsy) . .	1	
Pleurisy, with probable kidney disease . . .	1	Casts and kidney structures in the sediment of the urine.
Heart disease (hypertrophy, and valve affection) .	4	Kidneys healthy in three cases of p.m. examination.
Hemiplegia, from cerebral softening . . .	1	Blood corpuscles.
Pancreatic disease, causing icterus . . .	1	Amount of albumen very small.
Purpura hæmorrhagica . .	1	Case not fatal; nothing definite known about kidneys.
Typhoid fever	2	Blood casts and establishment of decided M. Brightii consecutive to the fever
Total	39	

2. The greater the quantity of albumen, the more likely is the existence of renal disease; and a "large" quantity of albumen ($\frac{1}{2}$ and upwards) is rarely found except in undoubted acute or chronic Bright's disease. It is necessary, however, in considering the amount of albumen, not only to have regard to the proportion in a particular specimen examined, but also to the total quantity in the twenty-four hours. This may be surmised by the density of the urine—low density indicating that the quantity of urine passed in twenty-four hours is large, and high density the contrary—but judged more accurately by ascertaining what is the actual flow of urine in twenty-four hours. A urine may be only slightly albuminous, but if it be of low density (under 1012) and the daily quantity between three and four pints,

the total loss of albumen will be very considerable, and the existence of renal disease strongly indicated. Indeed, of all urines there are none more surely indicative of Bright's disease than a pale, dilute, abundant urine which is, at the same time, more or less albuminous. On the other hand, as a rule with very few exceptions, when the urine is only slightly albuminous, and at the same time dense, and high coloured, Bright's disease is not present, and the albuminuria is owing either to pyrexia or to some impediment to the circulation of the blood.

The kinds of deposit which indicate most strongly the existence of organic renal disease are, (*a*) very abundant ones, containing casts and much renal epithelium; (*b*) those containing numerous casts and cells in a state of fatty degeneration. The least indicative of primary renal disease of serious import, are, blood casts, and very transparent casts in scanty numbers.

3. When the urine is found albuminous, and there exists neither pyrexia nor thoracic disease, or other recognisable condition which can account for the albumen, the inference is almost irresistible that there exists a primary organic disease of the kidneys.

XI.—SUGAR IN URINE.

In 1862 Schunck[*] announced that, when healthy urine was subjected to boiling with acids, it gradually deposited a resinous substance, and acquired the power of reducing the oxide of copper—in other words that the presence of a substance having the properties of glucose became apparent in it. This important observation probably explains the discrepant conclusions of those who have sought for sugar as a normal constituent of healthy urine. Brücke[†] and Bence Jones were always able to obtain sugar from healthy urine in not inconsiderable quantities. Bence Jones* obtained as much as 0·8 to 1·7 grain per pint. If natural urine contain a substance, capable of yielding sugar by a simple decomposition, it is quite possible that the sugar found by these observers was, either partly or wholly, an educt of the analysis, and not a pre-existing constituent of the urine. This much is certain, that healthy urines, and the vast majority of morbid urines, do not contain sugar in quantity capable of being detected by the most delicate direct testing. At the same time it is not improbable that minute traces of sugar, as of nearly every other substance dissolved in the blood, may exist in the urine. These traces, however, granting them to exist, have no clinical significance whatsoever. When sugar is present in quantity sufficient to interest the medical practitioner, it is detectable with certainty by direct testing; and conversely when direct testing reveals the presence of sugar it is invariably a grave pathological sign, and not a matter of mere physiological curiosity. In the following observations I have solely in view sugar in these sensible proportions.

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[*] Philosophical Magazine, March, 1862.

[†] Iwanoff has pointed out some fallacies in Brücke's process. He considers that the greater part of the sugar obtained by Brücke did not pre-exist in the urine, but was derived from some other constituent (indican) by the reagents employed. Iwanoff concludes that minute traces of sugar do exist frequently, but by no means constantly, in healthy urine. (Meissner's Bericht in Henle and Pfeufer's Zeitsch. for 1861, p. 323). In Bence Jones's process sulphuretted hydrogen was employed instead of oxalic acid; but, even with this modification, the urine would be rendered acid, and there would be great probability of sugar being produced from indican during the long process of evaporation of the large quantities (1000 c. c.) of urine used.

Tests for Sugar in Urine. (*Qualitative testing*).—Frequent mistakes are committed in regard to the presence or absence of sugar in urine, not only by physicians and surgeons, but even by professed chemists. More than once, specimens have been brought to me with the statement that an analytic chemist had found a small quantity of sugar, but in which no sugar really existed—certain fallacious appearances, to which I shall presently refer, having been mistaken for genuine evidence. Without proper precautions, sugar-testing, like all other testing, is open to fallacies; but with moderate care and observance of a few fixed rules, the detection of sugar is a matter of the most perfect certainty and of exquisite delicacy. Before proceeding to describe the *best* means for this purpose, I will say a word about those tests which are in common use, but which are either unreliable or insufficiently delicate — namely, the growth of torulæ, Moore's test, and the fermentation test.

1. *The growth of torulæ.* — Dr. Hassall has advanced the opinion that the yeast plant only grows in saccha-

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* Journal of the Chemical Society, 1862, p. 22.

rine urine, and that consequently the appearance of this fungus is positive proof of the presence of sugar. But as already stated, the yeast plant may be found growing to its full development in urines (practically) perfectly free from sugar. Moreover, it is not easy to discriminate the sporules and filaments of the yeast plant from those of the common mould (penicilium glaucum), and as both plants are far more frequently met with in an immature condition than in the state of fructification, the practical value of this mode of detecting sugar is reduced to insignificance.

2. *Boiling with liquor potassæ* (*Moore's test*).—When urine containing sugar is boiled with an equal bulk of liquor potassæ, the mixture darkens, and eventually assumes a brandy-brown colour. From its easy application, this test, as a preliminary step, and for negative evidence, is of great convenience. It has, however, two faults—(*a*) it is wanting in delicacy, and (*b*) it is liable to a notable fallacy. Moore's test does not answer clearly until the proportion of sugar rises to about 0·3 per cent. or one grain and a half to the ounce. By-and-by we shall come to a test *twelve* times more delicate than this.

Again, all high-coloured urines of high density become darker when boiled with liquor potassæ, although free from sugar; and albuminous urines, even when not high-coloured, darken sensibly under the same treatment. This occurs with perfectly fresh liquor potassæ; but if the test have been kept in ordinary white glass bottles, it very speedily becomes impregnated with lead, which it attracts from the glass, and this offers an additional source of error. The liquor potassæ kept in the wards of the Royal Infirmary was found largely impregnated with lead, although it had not been in use more than about six weeks. Liquor potassæ thus vitiated, when boiled with certain urines, turns them of a dark porter-brown colour. This is something quite different from the slight deepening of the tint which has been just alluded to, and it only occurs in *albuminous* urines, and not even in all of these. In acute Bright's disease, especially when there was blood in the urine, or when the albumen was abundant and associated with free discharge of renal epithelium, the change of colour was most intense; and in one such case the existence of sugar had been inferred therefrom, and announced to the patient and his friends, by the medical attendant. Where the proportion of albumen was small, and renal desquamation slight, the lead-tainted liquor potassæ did not produce so marked an effect. In such urines a slight darkening of colour only ensued, much to the same degree as occurred with fresh liquor potassæ. It was never found that liquor potassæ containing lead produced a dark brown coloration with non-albuminous urines, provided, of course, that they were sugar-free. The usual slight deepening of the tint took place, but not anything conspicuously greater than with fresh and pure liquor potassæ.

3. *The fermentation test.*—When saccharine urine is mixed with yeast and kept in a warm place, it speedily ferments with the production of alcohol and evolution of carbonic acid; and as no other substance is capable of undergoing this transformation, the occurrence of fermentation with yeast is certain proof of the presence of sugar.

Applied to ordinary diabetic urine, fermentation affords very clear indications. The most convenient and elegant way of applying it is the following:—A few crumbs of German yeast are put into the bottom of a test-tube; this is then filled up to the brim with the suspected urine, covered with an evaporating dish or saucer, and then inverted. The dish and inverted tube are now set aside in a warm place—say on the mantel-piece. The urine soon begins to ferment, gas collects in the top of the inverted tube and expels a portion of the urine; and if sugar be abundant, the gas accumulates in such quantities that all the urine is driven out before it. There is a precaution, however, to be observed. Some specimens of yeast spontaneously evolve bubbles of gas; it is therefore desirable, where the indication is doubtful, to perform a parallel experiment with the same yeast mixed with simple water, so that the amount of gas spontaneously yielded by it may be ascertained. German yeast is exceedingly convenient for fermentation experiments, and it has now come into such common use that a pennyworth may be purchased in almost any baker's shop.

There are two drawbacks to the clinical application of this test—(*a*) it takes some hours for its accomplishment, and (*b*) it does not suffice for the discovery of minute quantities. Urine is capable of absorbing somewhere about its own bulk of carbonic acid, so that unless the amount evolved be greater than this there will be no accumulation of gas in the top of the tube, and consequently no visible sign of fermentation. According to my experience, urines containing 0·5 per cent. or two grains and a half to the ounce and under, yield no sign to the fermentation test. Fermentation is therefore a considerably less sensitive method of sugar-testing than Moore's plan of boiling with liquor potassæ.

There is, however, another manner of applying fermentation to the detection of sugar, which is much simpler and even more delicate than the foregoing—namely, by comparison of the specific gravity of the suspected urine *before* and *after* fermentation. This proceeding will be examined more in detail under the head of quantitative testing; but I may here observe that considerably less sugar than one per cent. may be detected by the lowering of the density after fermentation.

4. *Reduction tests.*—The action of grape sugar on a number of metallic salts is attended with a reduction of the oxides which they contain to a lower degree of oxidation, or to the metallic state. Accordingly some of these salts are resorted to as valuable sugar tests, both qualitative and quantitative. The salts best adapted for this purpose are those of copper, bismuth, silver, chromium, and tin; but as the oxide of copper is the most universally known, and with proper precautions the most striking and sensitive, I shall confine my remarks solely to it.

The ordinary mode of using the copper, or, as it is called, *Trommer's test*, is to add a drop or two of a solution of sulphate of copper to the suspected urine in a test-tube. Liquor potassæ is then added in excess, and the mixture boiled. If the proper proportions have been observed, a red deposit of suboxide of copper falls when sugar is present. Applied in this rough way the operation of the test is very unsatisfactory. If the copper be in excess, a quantity of the protoxide remains undissolved and causes confusion. The liquor potassæ likewise obscures the result by producing an intense dark brown colour if sugar be abundant, and the boiling continued beyond a few seconds. In consequence of these and other objections, Trommer's test is regarded with very little favour by many practitioners, who rely in preference on the easy and ready, though less delicate, method of boiling with liquor potassæ. But all the uncertainty attending the employment of the copper test arises from a faulty application, and not from inherent imperfection. When skilfully used, it possesses a delicacy and certainty that renders all other reagents superfluous.

The first necessity is to abandon the rough method above sketched, and to prepare before-hand a test solution which shall combine the copper and the alkali in due proportion. This is accomplished by dissolving sulphate of copper in strong liquor sodæ with the aid of tartrate of potash. The exact formula for this solution (Fehling's standard copper solution) is given at p. 139.

Having prepared the test-fluid, it is employed in the following manner:—Fill a test tube to the depth of three-quarters of an inch or so with the copper solution; heat until it begins to boil, and then add *a drop or two* of the suspected urine. If it be ordinary diabetic urine, the mixture, after an interval of a few seconds, will turn *suddenly* of an intense opaque-yellow colour, and in a short time an abundant yellow or red sediment falls to the bottom. If, however, the quantity of sugar present be small, the suspected urine is added more freely, *but not beyond a volume equal to that of the test employed.* In this latter case it is necessary to raise the mixture once more to the boiling point. It is then allowed to cool slowly. If no suboxide has been thrown down when it has become cold, then the urine may with certainty be pronounced sugar-free.

The points of importance in this proceeding are—(*a*) to boil the test first, and not the urine; and (*b*) to use an excess of the test.

The first point is of importance, because the test-solution is apt to deteriorate by keeping, unless preserved hermetically sealed from the air. When deteriorated by exposure to the atmosphere, a deposit of suboxide takes place from it on simple boiling. Boiling the test, therefore, is a trial of its perfection. If it remain clear for a minute or two after ebullition, the solution is in order, and the testing may be proceeded with; but if the solution become somewhat opaque, and a red deposit presently fall from it, this deposit must be first filtered from the clear fluid, which is thereby again rendered fit for use: or—which is indeed the better plan—a fresh supply of the test is prepared. The deterioration here spoken of arises from the conversion of a portion of the tartaric acid into racemic acid, which, equally with sugar, has a reducing power on the oxide of copper, and when present, of course corrupts the analysis.

The necessity for using an excess of the test applies equally to an ordinary diabetic urine, as well as to one which contains only a small proportion of sugar and has a composition approaching the natural standard; but as the reason for employing an excess is not the same in the two instances, and as there are important differences in the operation of the test in the two classes of urine, I shall call attention to them separately.

(*a*) *Method of testing ordinary diabetic urine.*—Practically, the urine of a diabetic patient, where the disease is in full career, may be regarded as a solution of grape-sugar in simple water. The natural constituents are in such small proportion, owing to the increased flow, that they do not sensibly interfere with the operation of the test.

If, after the test has been heated to ebullition, *one* drop of diabetic urine be added, the reaction occurs almost instantaneously, and the suboxide falls of a brick-red colour at once; but if *several* drops of the same urine be added, the precipitate is a rich yellow. This difference in colour is merely a question of excess or deficiency of the test. When the copper exceeds the sugar, and the solution still retains its blue colour, the suboxide falls red; but if the sugar exceed the copper, and the blue colour have disappeared, the suboxide falls yellow.

The common mode of proceeding—that is, boiling the urine first, and then adding the reagent—is very objectionable, inasmuch as it may betray the operator into a too sparing use of the test, and thereby entail a failure of the reaction. If the sugar preponderate greatly over the copper, *no precipitation whatever ensues*, because the excess of sugar dissolves the suboxide, and forms with it a transparent yellow solution. This statement may be readily verified by boiling some diabetic urine in a test-tube, and then dropping in the test-solution. The first few drops occasion a dense, muddy, yellow opacity in the topmost layer; but when the tube is shaken the precipitate is redissolved. On adding more of the test, however, the opacity becomes permanent, and an abundant deposit presently subsides.

(*b*) *Method of testing where the quantity of sugar is small and the natural constituents approximate their usual proportions.*—The discovery of sugar in such a urine is much more difficult than in the former case. The ordinary ingredients of the urine—urea, uric acid, pigmentary and other extractives, the alkaline and earthy salts—seriously affect the delicacy of the test. If grape-sugar be dissolved in simple water, such is the sensitiveness of the reaction that one grain in ten pints yields a perceptible deposit; but when dissolved in urine, a considerably larger quantity may be present and the test fail to reveal its existence. Nevertheless, enough of delicacy still remains to satisfy all the requirements of clinical research.

Urine of the kind here considered—with a minute proportion of sugar, and the ordinary ingredients almost natural—is met with in the early stage of diabetes, before the disease has acquired its full development; also in convalescence from the less severe forms; and not unfrequently towards the fatal close of the complaint. Even in well-marked diabetes there are conditions under which the urine temporarily returns nearly to its natural state. These are—abstinence from saccharine and amylaceous food, and, *à fortiori*, abstinence from all food; accordingly, the morning urine, after the prolonged fast of the night may, in the less severe cases, be found almost sugar-free. A like effect follows the advent of an intercurrent inflammation, as of the lungs or lining membrane of the bowels.

In testing for sugar in urines of this description certain precautions are rigidly demanded, otherwise considerable quantities of sugar may be wholly overlooked. The most important of these is to use a great excess of the test. When the copper solution is added drop by drop to healthy urine, at a boiling heat, the blue colour is immediately discharged, although not a particle of sugar be present, and the urine assumes a deep-amber tint. The degree to which urines exercise this decolorising property varies with their strength—that is, with their concentration. A dense urine (sugar-free) will discharge the colour from nearly its own bulk of Fehling's standard solution; but even the most dilute natural urines—those that are almost colourless—have a very considerable power this way. Whatever be the nature of the transformation here involved, it is certain that when the colour of the test has been thus discharged, the copper it contains is no longer capable of being precipitated by any sugar that may be present in the urine; and the suboxide is not thrown down until such an amount of the solution has been added that the mixture retains a distinctly green tint after being raised to the boiling point. To secure an excess of the test the most certain method is to heat the solution first, as already recommended, and to add the suspected urine afterwards. Another advantage is secured by this proceeding. When the suspected urine contains a considerable quantity of earthy phosphates, the precipitation of these by the alkali of the test is apt to cause embarrassment. The phosphates fall in light, dirty-white flocculi, which might be mistaken by the unwary for a deposit of suboxide. When the test and urine are mixed together before applying heat, or the test is added to the boiling urine, the earthy phosphates fall in such fine flakes that the transparency of the mixture is impaired; but if the urine be added to the boiling test, the mixture retains its translucency from the phosphates being thrown down in denser masses; and by holding the tube between the eye and the light, the flakes are seen floating in a clear, blueish-green medium.

In the class of saccharine urines now under consideration, the suboxide is always precipitated yellow, never red. The operation of the test is exceedingly distinctive, and takes place as follows:—The copper solution having been heated to ebullition, and something less than an equal bulk of the suspected urine having been added, the mixture is again raised to the boiling point. It then changes to an intense opaque yellowish green, and slowly a bright yellow deposit subsides. If the urine contains less than half a grain per cent. of sugar, the precipitation does not take place immediately, but occurs as the liquid cools—in five, ten, or twenty minutes, and the manner of the change is peculiar. First, the mixture loses its transparency, and passes from a clear olive-green to a light greenish opacity, looking just as if some drops of milk had fallen into the tube. This green milky appearance is quite characteristic of sugar. By this proceeding one-tenth of a grain per fluid ounce, or less than one-fortieth of a grain per cent., can with certainty be detected, and any quantity below this has no pathological signification, and is matter of only physiological interest.

Some of the natural urinary ingredients, and especially uric acid, have been stated to possess the power of reducing the oxide of copper to a state of suboxide, and of becoming thereby the source of a notable fallacy in using this test for the detection of sugar. No fear, however, need be entertained on this score ; I have over and over again treated urines containing an excess of uric acid, and even urines thick with the amorphous urate deposit, with the test solution at a boiling heat, but have never obtained the least resemblance to the sugar reaction. It is, however, to be borne in mind, that if urine be boiled with the test *for a considerable time*—say twenty minutes or half an hour—a reddish deposit falls, and the mixture assumes a muddy, dirty-fawn appearance, although no sugar be present. The reddish deposit appears to consist of the earthy phosphates tinged red by some of the suboxide, reduced, perhaps, through the instrumentality of uric acid. But this reddish deposit is *only produced after prolonged boiling*, and prolonged boiling is of all things the most to be avoided, because the most utterly useless, in performing the test. If simply raising the fluid to the boiling point, and then allowing it to cool in a warm place, as in a jug of hot water or on the hob, fail to yield an indication of sugar, no amount of boiling will develop a trustworthy reaction.

To recapitulate: the best method of detecting sugar in urine is as follows:—Pour some of the prepared test-liquor into a narrow test-tube to the depth of three-quarters of an inch ; heat until it begins to boil ; then add two or three drops of the suspected urine. If sugar be abundant, a thick yellowish opacity, and deposit of yellow suboxide are produced (and this changes to a brick-red at once if the blue colour of the test remain dominant). If no such reaction ensue, go on adding the urine until a bulk nearly equal to the test employed has been poured in; heat again to ebullition: and, no change occurring, set aside without further boiling. If no milkiness is produced as the mixture cools, the urine may be confidently pronounced free from sugar, for no quantity above a fortieth of a grain per cent. can escape such a search, and any quantity below that is devoid of clinical significance.

Estimation of the quantity of sugar in urine (*Quantitative testing*).—In early times medical men judged of the quantity of sugar in diabetic urine by the amount of syrup yielded on evaporation. This was a very rude as well as troublesome proceeding. A much readier and not less precise method was to calculate the sugar from the specific gravity. Dr. Henry drew up a table, which Prout afterwards extended and improved, showing at a glance how much solid matter per pint was contained in urines at different densities. When the urine voided amounts to several quarts a day, and the natural urinary ingredients have sunk to a very low proportion, the secretion resembles a solution of grape-sugar in pure water. In this condition the density is a moderately accurate measure of the quantity of sugar; but it is still far from absolute correctness, as may be judged from the following table, drawn up from a number of my analyses:—

TABLE *showing the uncertain relation of the specific gravity to the proportion of sugar where the daily flow of urine ranged between nine and thirteen pints.*

Specific gravity.	Sugar per imperial pint.
1045	875 grains.
1043	972 „
1042	683 „
1041	920 „
1041	931 „
1040	911 „
1039	683 „
1035	875 „
1034	645 „
1033	635 „

But when the flow of urine is no more than two or three pints a day, the natural ingredients come to hold something like their normal proportions, and contribute very sensibly to raise the density. Accordingly with the diminished flow there is a very greatly lessened proportion between the specific gravity and the per-centage of sugar. The annexed table shows this relation in the urines of the same patients when the daily excretion had been reduced by dietetic means to *between two and three pints.*

TABLE *showing the lessened and still more uncertain relation of the specific gravity to the quantity of sugar where the daily flow ranged between two and three pints.*

Specific gravity.	Sugar per imperial pint.
1044	625 grains.
1042	553 ,,
1041	591 ,,
1041	498 ,,
1039	568 ,,
1039	608 ,,
1039	600 ,,
1039	446 ,,
1036	377 ,,
1035	471 ,,
1034	486 ,,
1034	312 ,,

On comparing these two tables, it is seen that the density holds a much less constant relation to the proportion of sugar when the daily flow is scanty than when it is abundant. It is also seen that in the former case a given degree of density indicates a much lower proportion of sugar than in the latter. The mean density in the first table is 1039·3, and in the second nearly the same—1038·6 ; but the proportion of sugar is much greater in the first, where it averages 813 grains per pint, than in the second where it is only 511 grains.

Of the more accurate methods there are two peculiarly eligible for practical use—the one on account of its speedy performance, and the other on account of its easy application.

1. *Volumetrical method.*—This has been brought to a high state of perfection by Fehling. It depends in principle on the fact that there is a fixed relation between the amount of a copper salt reduced to a state of sub-oxide and the sugar present. Fehling found that one equivalent of grape-sugar, or

180 parts, decomposed exactly ten equivalents, or 1246·8 parts, of sulphate of copper. Accordingly he prepared a solution of copper of standard strength, and applied it to fluids containing grape sugar; and the quantity of these required to decompose a fixed volume of the standard solution furnished an exact measure of the sugar they contained.

Fehling's standard solution is prepared according to the following prescription :*—

Sulphate of copper, 90¼ grains ;
Neutral tartrate of potash, 364 grains ;
Solution of caustic soda, sp. grav. 1·12, *four* fluid ounces ;
Add water to make up exactly *six* fluid ounces.

200 grains of this solution are exactly decomposed by one grain of sugar. The apparatus required for the performance of the analysis are described and figured at pp. 5 and 6.

Mode of performing the analysis.—Measure off 200 grains of the standard solution in the 200-grain tube, pour this into the flask, and add about twice its volume of water; then place over a spirit-lamp to boil. While the copper solution is being heated, the urine to be analysed should be diluted with water to a known degree. In the case of ordinary diabetic urines, the best dilution is one in ten. This is obtained by carefully filling the 6-oz. measure with water to the depth of 4½ ounces, and then adding urine so as to make up exactly 5 ounces. The mixture will then contain exactly one-tenth of urine. (When the quantity of sugar in the urine is very small, a dilution of one in five, or even the undiluted urine may be employed.)

The next step is to fill the burette (which is graduated to grains) with the diluted urine to 0. Then proceed to add it, in successive small portions, to the boiling copper solution, until the blue colour has entirely disappeared. After each fresh addition from the burette the mixture should be raised to the boiling point, and then allowed to stand a few seconds, so that the precipitated copper may subside, and the observer may see, by holding the flask between the eye and the light, whether the mixture still retains any blue colour. As soon as the blue colour has disappeared the analysis is complete, and the quantity of diluted urine employed may be read off. The percentage of sugar in the urine can now be readily calculated. Suppose 125 grains had been added from the burette: this represents one-tenth, or 12·5 grains, of undiluted urine, and contains exactly one grain of sugar: by dividing 12·5 into 100, the per-centage of sugar is obtained: or $\frac{100}{12 \cdot 5}=8$: the urine contains 8 per cent. of sugar.

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* More exactly, in grammes and cubic centimètres, the proportions stand as follows :—

40 grammes—crystals of sulphate of copper ;
160 grammes—neutral tartrate of potash ;
750 grammes—caustic soda, sp. grav. 1·12.
Add water up to 1154·5 cubic centimètres.

Each 10 cubic centimètres correspond to 0·05 gramme of grape-sugar.

2. *Differential density method.*—The second of the two methods of estimating sugar, which I have undertaken to explain, combines, as I believe, more perfectly than any other, the twin advantages of ease and accuracy. It is founded on the diminution of density suffered by saccharine urine when fermented with yeast. The specific gravity of an ordinary diabetic urine ranges from 1035 to 1050. When it has undergone fermentation, and all the sugar is converted into alcohol and carbonic acid, the specific gravity is found to have sunk to 1009, to 1002 or even below 1000. This falling off in the density arises from two distinct yet necessarily associated causes—namely, first, the destruction of the sugar, which was the cause of the high density of the original urine; and, second, the presence of the generated alcohol in the fermented product. Now, the loss of density from these causes must evidently stand proportional to the quantity of sugar originally present in the urine, and must consequently furnish a measure of its quantity.

The experimental data on which this method is founded are fully detailed in a paper published by the author in the "Memoirs of the Manchester Literary and Philosophical Society" for 1860; also in a paper in the "Edinburgh Monthly Journal" for October, 1861. The mode of experimenting was—first to ascertain by the volumetrical analysis, which I have just described, how much sugar was contained in a certain diabetic urine. The urine was then fermented by means of

German yeast—its specific gravity having been previously ascertained. In twenty-four hours, after the fermentation had ceased and the scum had subsided, the density was taken again, and by subtracting this from the density before fermentation, the "density lost" was ascertained. And it was found that for every grain of sugar contained in an ounce of urine, one degree of specific gravity had been lost. Experiments were multiplied on diabetic urine; corresponding experiments were made with solutions of sugar of known strength in healthy non-saccharine urine and in pure water, and the issue of all was to establish the conclusion that *the number of degrees of "density lost" indicated as many grains of sugar per fluid ounce.*

In the practical application of the method, the ordinary urinometer may be used for taking the densities; but it is well to choose one with a long scale, as some of those in use have very short ones, and it becomes impossible to read the density accurately. Still further precision may be attained by dividing the usual scale into two parts on separate instruments. I have had constructed for my own use two perfectly corresponding urinometers, on one of which the scale ranges from 995 to 1025, and on the other from 1025 to 1055, each instrument covering 30 degrees of density. The scales are thus rendered so long, and the intervals between the lines so great, that in a clear urine the specific gravity can be easily read to a quarter of a degree; and even in the fermented urine, which does not regain its original transparency, but continues, at least for many days, more or less cloudy, it can be read with certainty to half a degree.

Another important point is to obviate errors from variations of temperature. If the density before and after fermentation be taken at widely different temperatures, an error of serious amount may creep into the analysis. The best mode of avoiding this is to put up a few ounces of the unfermented urine in a "companion phial," and to place this side by side with that set apart for fermentation, so that, at whatever temperature the fermented product may be when its density is observed, its unchanged *alter ego* stands near it for comparison at exactly the same temperature.

The most convenient way of proceeding is the following:—

About four ounces of the saccharine urine are put into a 12-ounce bottle, and a lump of German yeast about the size of a cobnut or small walnut is added to it. A great excess of yeast is used to hasten fermentation, but a little more or a little less does not sensibly affect the result. The bottle is then covered with a nicked cork (which permits the escape of the carbonic acid), and set aside on the mantelpiece or other warm place to ferment. Beside it is placed a tightly-corked 4-ounce phial filled with the same urine without any yeast. In about twenty-four hours the fermentation will have ceased, and the scum cleared off or subsided. The fermented urine is then decanted into a urine-glass, and its specific gravity taken; at the same time, the density of the unfermented urine in the companion phial is observed, and the "density lost" ascertained. Fermentation is generally complete in about eighteen hours if the locality be sufficiently warm; and it is desirable to remove the two phials into a cool place two or three hours before the densities are taken, in order that they may attain the temperature of the surrounding atmosphere.

The two following examples may serve as illustrations of the method:—

	I.	II.
Density before fermentation . . .	1053	1038
Density after fermentation . . .	1004	1013
Degrees of density lost . . .	49	25
Grains of sugar per fluid ounce . .	49	25

If it be desired to bring out the result as so much per cent., this is accomplished by multiplying the number indicating the "density lost" by the coefficient 0·23. Thus in the first of the above examples 49 × 0·23 = 11·27, and in the second 25 × 0·23 = 5·69, which are the amounts of sugar respectively per 100 parts.

The time actually consumed in determining the quantity of sugar in urine by this method does not exceed four or five minutes, but the result must be waited for until the succeeding day; this is its chief disadvantage. Its application is so easy, that a medical friend in attendance on a diabetic patient, was able to teach the patient's wife to make the analysis; every morning when he came, she could give exact information as to the quantity of sugar excreted on the previous day.

Optical saccharimetry.—The property of glucose of rotating the plane of polarisation to the right has been taken advantage of to estimate the quantity of sugar in diabetic urine. The best instruments for the purpose are those of Mitscherlich and Soleil. This method is not so universally applicable as the two preceding; and the price of the instruments, together with the delicacy required in their manipulation, puts them almost out of reach of ordinary practitioners.

Clinical significance of sugar in the urine.—The presence of a large quantity of sugar in the urine is the characteristic feature of diabetes mellitus: but small quantities may be present in a variety of other circumstances—as after eating excessively of amylaceous or saccharine articles of food, from injury or disease of certain parts of the nervous system, from impediments to respiration, etc. This subject, however, can be more conveniently treated in a future page (see PHYSIOLOGICAL CONSIDERATIONS RELATING TO DIABETES).

PART II.

URINARY DISEASES—DISEASES OF WHICH THE CHIEF CHARACTERISTIC IS AN ALTERATION OF THE URINE.

CHAPTER I.

DIABETES INSIPIDUS.

WILLIS—Urinary Diseases, p. 1. Lond. 1838.
FALCK—Beitr. z. Lehre von d. Einfache Polyurie. Deutsche Klin. 1853.
NEUFFER—Ueber D. Insip. Tubingen Thesis, 1856. Canstatt's Jahresb. 1857, iv. 234.
TROUSSEAU—Clinique Médicale, T. ii. p. 611.
MAGNANT—Du Diabète Insipide. Strasburg Thesis, 1862.
P. EADE—On Diabetes Insipidus. Beale's Archives, vols. ii. and iii.
W. STRANGE—Case of Diab. Insip. Beale's Archives, vol. iii.
ANDERSOHN—Nichtzuckerfürender Harnruhr. Dorpat Thesis, 1862.

CASES characterised by increased thirst and excessive discharge of a watery urine of low specific gravity, free from sugar and albumen, are grouped together under the general designation of diabetes insipidus.

The want of uniformity in the course and symptoms of these cases, and in the anatomical changes found after death, indicate that several wholly distinct pathological states are included under this heading.

Attempts have been made to classify the cases according to the characters of the urine. Those in which it was supposed that the urine merely contained an excessive amount of water, without any alteration of the total quantity of solids excreted, or of the mutual proportion of the several solid ingredients to each other, have been named *Polydipsia* (or excessive thirst); those in which it was supposed that the solid matters, and especially urea, were excreted in excessive quantity, have been named, *Polyuria;* and those in which it was supposed that the urea and other solids were in diminished quantity have been named *Anazoturia* (Willis).

This classification is, however, valueless in practice: both from the difficulty of assigning a precise standard of composition to the urine under the various conditions of existence, and the tedious and difficult investigations, extending over several days, which are required to ascertain the mean composition of the urine in any particular case.

I have collected from various sources, most of which are indicated at the head of this chapter, sixty-six cases of insipid diabetes; to these I have added three cases observed by myself; and three more, of which the particulars were supplied by my friends Drs. Bates and Bowman. From an analysis of these seventy-two cases the following account has been drawn up.

Etiology.—The liability to diabetes insipidus is very considerably greater in males than females; of the seventy-two cases, fifty-two were males, and twenty females. The *age* of the patients at the time of invasion ranged from the extremes of infancy to old age; but the greater number occurred between the ages of five years and thirty years. In the following table an analysis is given of the ages of sixty-five cases at the time of invasion.

Infancy	7 cases.	From 20 to 30 years	15 cases.
From 5 to 10 years	12 "	" 30 — 50 "	14 "
" 10 — 20 "	13 "	" 50 — 70 "	4 "

In two, if not three, cases the disease appeared to have existed actually from birth.

In a very large proportion, no *exciting cause* whatsoever could be assigned for the disorder. In the remainder, various circumstances were alleged with greater or less probability to have been the exciting causes. These present considerable similarity to the alleged causes of saccharine diabetes, and stand in the following order of frequency:—

Blows on the head, and falls	6	Previous febrile or inflammatory disease	4
Cerebral disease (tubercle, &c.)	5	Hereditary influence	3
Intemperance	5	Muscular effort	2
Exposure to cold and drinking cold fluids while heated	4	Exposure to hot sun	1
		Mental emotion	1

In five cases serious organic changes were found in the kidneys. These will be more particularly described in connection with the morbid anatomy of the disease.

Two cases recorded by Dr. W. Watts (Lancet, 1848) are referred by him to syphilitic disease and abuse of mercury.

Hysteria, grief, neuralgia, or the influence of a nervous constitution, are also mentioned as determining causes.

In some of the traumatic cases the symptoms set-in with maximum intensity on the very day of the accident ; in others there was at first loss of consciousness ; and the thirst and diuresis came on with the restoration of the faculties, or a few days after. In one case severe nervous symptoms continued for six months after a fall, and the diuresis first broke out at the end of this period. In four of the traumatic cases the symptoms persisted for between nine days and a month, and then finally disappeared as the cerebral symptoms subsided ; in two others the disorder became permanent, and had already existed at the date of the record, six years in one and seven years in the other.

Of the five cases attributed to spontaneous cerebral disease, only one (observed by myself, and detailed further on) was examined *post mortem*. In this, tubercles were found in the brain. In another, also observed by myself (a shopkeeper, thirty-five years of age), the disease had come on twenty months previously with sudden, complete, and permanent loss of sight, first in the left eye, and six months later in the right. During these twenty months the patient had been in the habit of voiding two or three gallons of urine daily. He was also subject to curious nervous attacks, which recurred at irregular intervals, and lasted from half an hour to periods of several days. They consisted in a perversion of intellect, incoherence, irrepressible impulse to go away from the house, trembling of the limbs and twitching of the muscles. Sometimes the patient would fall into an epileptiform fit, with loss of consciousness, screaming and convulsions, but without foaming at the mouth, or biting the tongue. When seen by me he was totally blind, but the intellect was perfect, and the general health—except during the paroxysms—was good. He could walk twelve miles with ease; and in the last eight months he had gained weight to the extent of 40 lb. The history and general character of the symptoms appeared to point to the existence of vesicular parasites within the cranium. The three remaining cases were children supposed to suffer from cerebral tubercle. They all died in convulsions.

Of the five cases attributed to intemperance, the symptoms came on in one of them on the day after a severe bout of drinking, in which the patient had been insensible for two days. Not one of this group is reported as cured; and one died in two months.

Two cases followed exposure to cold; and two followed copious drinking cold fluids while the skin was hot and perspiring. One of the latter, related by Vigla, fell ill with unquenchable thirst and diuresis on the same day, and died a few months after.

Four cases followed variola, ague, fever, and inflammation of the bowels; all ran a very chronic course, and lasted from four to twenty-four years, with good preservation of health; the symptoms commenced immediately after recovery from the initial complaint.

In two cases, the symptoms commenced immediately after violent muscular effort. One was a boy of twelve, who strained himself in pushing a cart-wheel sunk in the mud. After a few months, the symptoms were subdued by nitrate of potash; but some months later a relapse occurred, and the patient died suddenly, from taking, as is alleged, too large a dose of the nitrate (P. Frank—cited by Romberg). The second is a remarkable case, related by Jarrold, in Duncan's Annals for 1801. A girl of 19, when going down a flight of steps, slipped: with very great exertion she saved herself from falling. Immediately after menorrhagia began, and on the evening of the same day she experienced inordinate thirst and profuse diuresis. She entered the Edinburgh Infirmary, under Professor Gregory, and was speedily cured of the hæmorrhage by the compound powder of alum. The urine amounted to the enormous quantity of 50 lb. in the twenty-four hours, sometimes even to 60 lb., and one day to 72 lb.! Under the influence of lime-water and powdered galls, the urine was gradually reduced to between 5 lb. and 10 lb. a day. She left the hospital otherwise in good health.

Three cases were attributed to hereditary influence. One of these was a man in good health who had suffered for the long period of fifty-nine years from polyuria. The disorder began in infancy. His father, two brothers, and a sister had suffered similarly. Another was a healthy soldier of twenty-four, who had been polyuric for four years. His mother, brothers, and two sisters suffered in the same way. The third was a young lady of nineteen, mentioned by Trousseau (Clinique Médicale, t. ii. 611), whose grandfather was affected with saccharine diabetes, and uncle with Bright's disease. She was well-grown and tolerably healthy, and had borne her complaint for six years. All these cases proved incurable.

Course and Symptoms.—The invasion of the complaint is often quite sudden. Dr. Bennett relates the case of a woman, thirty-four years of age, who went to her work one morning at six o'clock in her usual health; at eight o'clock, two hours after, she was suddenly seized with intense thirst and diuresis, which became persistent from that time.

In several instances it is recorded that an intercurrent febrile or inflammatory disorder temporarily suspended the symptoms. In one case, an attack of acute articular rheumatism (treated with nitrate of potash) suspended the disease permanently, after it had existed in intensity for eighteen years. In another instance (a girl of nineteen, polyuric from infancy), an attack of pleurisy was treated by a blister, which suppurated for thirty-five days; at the end of this time, both the pleurisy and the polyuria disappeared permanently.

The quantity of urine voided by persons afflicted with insipid diabetes, is usually considerably greater than in saccharine diabetes: 15, 30, and even 40 pints are frequently mentioned as the daily amount of urine. Its specific gravity varies from a little above that of pure water to 1003 and 1007. It is limpid and colourless, and contains but a feeble proportion of solid matters. The thirst is generally intense; often inextinguishable; in several cases the patients are stated to have drunk their own urine. When the quantity of drink and the quantity of urine were compared, sometimes the one and sometimes the other showed in excess. Careful determinations on this point by Falck, Neuschler, and others, indicate that if fluids be allowed *ad libitum* the urine voided is in about the same quantity as the drink; but if the imbibition of fluids be compulsorily diminished, the urine is not diminished in the same proportion, and dehydration of the tissues results.

The skin is generally dry and harsh; sometimes it preserves its natural moisture, and in rare examples sweating has been observed. It is noteworthy that boils and carbuncles are only once mentioned.

The state of the general health varies a good deal. In the greater number of the recorded cases fair health was preserved —in several patients the health was perfect, and some of them became fathers and mothers of families, and went about their usual avocations without other detriment than the inconvenience of a constant thirst and incessant calls to void urine. A remarkable example of this kind was communicated by Mr. Maxwell to Dr. Simmons (Med. Facts and Obs. vol. ii. 96). A hale farm labourer, aged fifty-one, who habitually performed the severest tasks, thrashing, mowing, etc., like his fellow-workmen, had been polyuric for twenty-four years. The disorder came on after a fit of ague. The patient drank daily, summer and winter, from 32 to 36 pints of water, and voided urine in proportion. Yet he slept well (except that he frequently awoke to drink): he had no pain or ache of any sort; he had an excellent appetite, a moist skin, and perspired freely when he was at work. Dr. Simmons also cites the case of a woman residing in Paris, who had been polyuric from infancy. In due time she married a cobbler, and became the mother of eleven children, of whom, however, only two were living when the case was recorded. Dr. Willis quotes the history of an artizan, aged fifty-five, who entered the Hôtel Dieu, of Paris, for some trifling bruise of the knee, from which he speedily recovered. From the age of five years he had suffered from a constant thirst, accompanied with a commensurate diuresis. From his sixteenth year he had drank on an average two buckets full of water daily. This man continued in good health; he was the father of several children, and experienced no inconvenience from his infirmity beyond what was inseparable from the frequent calls to pass water, and the constant necessity for drink.

This high state of health is however exceptional: more commonly the patients are very decided valetudinarians: and the symptoms from which they suffer bear a resemblance to those of diabetes mellitus, though rarely exhibited in equal severity. These are epigastric and lumbar pains; dry, harsh, hot skin; painful dryness and heat of the mouth and fauces; emaciation. Sometimes the appetite is voracious, more commonly moderate or indifferent. The temper is querulous: the mental faculties enfeebled; the bodily strength diminished; the sexual functions often abolished. The face is subject to erythematous congestion. Enforced abstinence from fluids aggravates most of these symptoms: the body then becomes unbearably hot, the skin suffused, a sense of intolerable sinking, or even of intense pain, is felt in the pit of the stomach, and the intellect becomes confused.

The loss of rest, the tormenting thirst, the mental worry, at length produce, in most instances, an exhaustion of the bodily vigour; œdema of the feet often appears towards the last; and death closes the scene.

In some cases there was dislike to vegetable aliments, in others to animal food. The cobbler's wife, before alluded to, was very sensitive to alcoholic drinks; a single glass of wine caused uneasy sensations in all her limbs, and a sense of faintness. In other instances the patients drank freely of wine or beer, as their condition allowed. In a man, observed by Trousseau, there was a remarkable tolerance of alcoholic stimulants. This man on one occasion drank a litre (a pint and three-quarters) of brandy in two hours; and while in hospital he imbibed daily a similar quantity without the smallest inconvenience. The patient related, that since his illness began he had acquired this singular immunity from the causes of drunkenness. More than once he had laid wagers to drink twenty bottles of wine at a single sitting, and had won his wagers without the least disturbance of the nervous system.

Irritability of the bladder, with excessively frequent micturition, was noted in several instances.

The *duration* of the complaint is exceedingly uncertain. The traumatic cases generally only lasted a few weeks or months: on the other hand, one of the congenital cases had endured fifty-nine years, another fifty years, at the date of the record.

Out of the seventy-two cases collected, sixteen are reported as complete recoveries; thirteen ended fatally; and the remaining forty-three were still in progress when reported; though, in some of them, considerable amelioration had taken place. In the sixteen recoveries the duration of the disease was comparatively short,—in nine, it was under a year; in one, four years; in two, eighteen and nineteen years; and in the remainder, "some" years. In the thirteen fatal cases, the duration was still shorter. In nine of them it was under a year; one died in the short space of seven weeks: two more in two months. The other four survived for periods varying from eighteen months to twenty years.

Of the forty-three cases still in progress, the duration of the disease is mentioned in thirty-one instances:—Four had continued for a year or under; five, for between one and two years; nine, for between two and six years; five, for between six and twelve years; four, for between twelve and twenty-four years; and four, for between twenty-four and fifty-nine years.

Morbid Anatomy.—The condition of the organs after death from diabetes insipidus, has only been ascertained in a few cases. I have found recorded five *post-mortem* examinations; and to these I add one performed by myself. In three of these cases the lesions found presented a tolerably close similarity, and consisted of an atrophied and degenerated condition of the renal substance; in a fourth, the glandular tissue of the organs was entirely wanting; in a fifth, multiple abscesses were found in the kidneys: in my own case, the kidneys were simply hyperæmic and somewhat enlarged, and notable disease was found in the brain.*

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* In an appendix to the present chapter reference is made to some cases of

As these cases are so few in number, I shall describe them more fully.

Case I. (Dr. Eade—Beale's Archives, 1861, p. 8.)—A man, aged sixty-five, had suffered from jaundice and neuralgia; he succumbed in eighteen months to the continual diuresis, and the urgent and incessant calls to void urine. The quantity of urine varied from three to six pints; specific gravity never exceeded 1008; it was free from sugar, albumen, or other morbid ingredient. The autopsy revealed the following: "the infundibula and pelvis of both kidneys were greatly dilated, and the state of sacculated kidney evidently in process of establishment. Left kidney of natural size. Right, one-half larger, and of darker colour. Both showed depressions along the surface, marking the interlobular portions. Previous to section, the cones could be distinctly felt as much denser than the inter-pyramidal portions, giving indeed the sensation of so many little tumours or nodules. On section, both were seen to be pale and flaccid, and evidently undergoing a gradual process of absorption." The bladder was somewhat large and thickened; the ureters dilated. The thoracic and the other abdominal organs were not diseased.

Case II. (Dr. Eade—Beale's Archives, 1862, p. 128.)—A man, aged sixty-two, had experienced excessive thirst and diuresis for twenty years. Health fair, until two years before death, when it began to fail, and for the last nine months he was unable to work. The quantity of urine often amounted to fourteen and sixteen pints, and had never contained sugar or albumen. There was little pain beyond a sense of weariness. The bowels were constipated, and the stomach very irritable with frequent vomiting. At length the bladder became unable to expel its contents, and a typhoid state supervened; the stomach rejected everything, and he died exhausted. *Autopsy.* Both kidneys were diminished in size, deeply lobed on the surface, and very dense to the feel in the position of the cones. On section they were seen to be greatly wasted. The cortical portions very thin, and scarcely to be distinguished from the pyramidal. The cones were nearly absent, or rather were converted into dense fibrous tissue, containing many large cystiform spaces. The mucous membrane of the pelvis was thickened, fibrous-looking, and darkly congested. The pelvic cavities considerably enlarged. Ureters a little dilated. On microscopical examination (by Dr. Beale) many of the tubes were found narrow and much wasted, while others were twice their natural diameters. The walls of the tubes were firm and thick. The capillary vessels everywhere were surrounded by a considerable quantity of fibrous material with numerous nuclei. The Malpighian bodies were, for the most part, smaller than in health. The epithelial cells were also smaller, as well as more numerous than in health, and the tubes appeared to be distended in many places by their accumulation. The supra-renal capsules were greatly diseased, and converted into flaccid cysts. The bladder was enlarged, and its walls thin and pale. The other abdominal organs were healthy, except perhaps the

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polyuria (with records of *post-mortem* examinations), in which a minute quantity of sugar existed temporarily in the urine.

liver, which was intensely congested. In neither case does the brain appear to have been examined.

Case III. (Neuffer—cited in Magnant's Thesis.)—A man, aged twenty-eight. The disease came on after a drunken bout. There was intense thirst; the urine amounted to thirteen or fourteen pints a day; specific gravity 1001 to 1002; without trace of albumen or sugar. He emaciated rapidly; had pain in the epigastrium; at length frequent vomiting; itching of the skin, which was dry; enfeebled vision. He died in about two months.

Autopsy.—The gastric mucous membrane was pale and swollen; the kidneys were notably diminished in size, pale, anæmic: the epithelium of the tubes fatty; bladder contracted, mucous membrane a little tumefied; other organs healthy.

Case IV. (Dr. Strange—Beale's Archives, 1862, p. 276.)—The patient was a farm labourer, aged eighteen, who presented the appearance of a moderately stout lad of fifteen. He was admitted into the Worcester Infirmary on October 19, 1861. The skin and tongue were natural, and the face ruddy; appetite normal; thirst constantly excessive; bowels generally relaxed. The urine amounted to about twelve pints in the twenty-four hours; its specific gravity was 1007; it contained neither sugar nor albumen. All the history obtainable was, that the patient had been a delicate and backward boy; that he had had this diuresis for a number of years, and that the medical attendant had always affirmed that the urine did not contain sugar.

Dr. Strange, being desirous to ascertain whether the diuresis was kept up by the excessive imbibition of fluids (in accordance with the theory of Professor Bennett and others), restricted the patient to a more moderate allowance of fluids. A warm bath was administered twice a week. Four days after admission (October 23) the urine measured nine pints; its specific gravity was 1006. On the 26th the bowels were much relaxed; urine five pints. On the 28th, a phosphoric acid mixture which he had been previously taking was omitted, and Mist. Cret. Co. given instead. On this day the patient complained for the first time of headache, with weakness and loss of appetite; there were also some febrile symptoms. On the 29th, the bowels being still relaxed, 5 minims of tinct. opii, and half a drachm of tinct. catechu were added to the mixture. On the 30th he became drowsy, with pain at the back of the head; the diarrhœa continued, with vomiting. Effervescing draughts, with nitric ether, were now administered in lieu of the previous medicines; half an ounce of brandy was given three times a day, and cold applied to the head. On November 2nd, the drowsiness and sickness had abated; the bowels were confined; the urine three and a-half pints, specific gravity 1004. The brandy was omitted, and half an ounce of castor oil administered. As it now appeared that restricting the patient in his drink had resulted in mischief, he was allowed to take as much water or barley-water as he pleased. On November 4th, in the morning, he was again drowsy; in the evening he was seized with convulsions, and shortly afterwards he became comatose and insensible, with dilated pupils and stertorous breathing. He was bled to ℥x, and much relieved thereby. The coma ceased, and consciousness and speech returned in a quarter of an hour. Mustard was applied to the feet, and a draught containing tinct. canthar, and sp. æth. nit., in camphor water, was given every third hour, with a view of restoring the accustomed diuresis. On the morning of the 5th he was conscious, and still had some headache. The diuretic mixture was continued, and a black draught administered immediately. On the 6th he was again found in a semi-comatose state, the pupils were dilated, and there was stertor, with sighing respiration. Six leeches were applied to the temples, mustard to the feet, and cold to the head. The coma became more profound, and he died at 9 p.m.

Autopsy.—The kidneys were found to be reduced to mere sacs, of from twice to thrice the extent of the healthy kidney. There was a complete absence of all proper parenchymatous substance, both tubular and cortical; the sacs being divided into a number of cells by the inter-tubular septa which occur in the fœtal state. The walls and septa were formed of strong fibrous tissue, lined with what appeared rather serous than mucous membrane, and the cavity and ureters contained a small quantity of the same urinous fluid which had been passed during life. The ureters were so much dilated that that on the right side was at first mistaken for the ascending colon. The circumference of the ureter varied from three to four and a-half inches. The kidney and ureter of either side were almost precisely in the same condition. The urine in the ureters and sacs was tested for urea by evaporation and nitric acid, without result. On closer examination no proper kidney substance could be discovered, nor did it appear that there ever had been any tubular or cortical portions; here and there were a few hard cartilaginous masses of very small size, closely adherent to the membrane forming the sac. The other abdominal and the thoracic organs were healthy. The brain was not examined.

Case V.—On the 29th of May, 1862, I saw, with Mr. J. Smith, of Stretford Road, a youth of sixteen years of age, who was passing a large quantity of a watery urine. He was moderately well-grown, exceedingly emaciated, weighing only 78 lb. Pulse 127; tongue glazed, red in the centre, and covered with a yellowish brown fur at the sides. The skin was dry and harsh. The patient was troubled with intense and incessant thirst, and voided from nine to twelve pints of urine daily. The appetite was bad. Neither the head nor chest were the seat of any subjective symptoms.

He gave the following account of himself. Previous to his present illness he was occupied as a clerk in a warehouse, and had enjoyed uninterrupted health until three months ago. About that time he noticed that he was getting thinner and weaker, that he drank a great deal, and never perspired. These symptoms had undergone a gradual and steady increase, and a fortnight ago had sustained an alarming aggravation. The patient could, nevertheless, still go about, and even take the air for short periods. He suffered no pain in any part, but he slept badly, and passed restless nights. The appetite had been indifferent from the very beginning, and it was now altogether lost. The bowels were moved almost daily, but there was a tendency to constipation. Dyspeptic symptoms—heaviness after food, flatulence, and occasional vomiting—had been noted from the commencement of the illness, but they did not attain a great severity at any time.

In searching back among the patient's antecedents for any determining cause, no fact of moment was elicited. The lad had been living in comfort, well-clad, well-fed, and well housed, with his grown-up sisters. No tuberculous or other family taint could be traced. The case had been treated with morphia, bismuth, and permanganate of potash, but with no result beyond a palliation of the dyspeptic symptoms.

The urine of the twenty-four hours was carefully collected and measured on six several occasions, and portions sent to me for examination. The characters of it were constant; it was pale like water, and the specific gravity varied from 1002·7 to 1004. The quantity was between nine and ten pints at the time of my visit. It afterwards increased to fourteen pints daily. There was neither albumen nor sugar in it, and its re-action was faintly acid. The quantity drank was found, on exact measurement, to be almost precisely equal to the quantity of urine. The amount of urea varied from 0·4 to 0·55 per cent., and from 394 to 505 grains in the twenty-four hours. This was an enormous quantity for the weight of the body. According to the mean results tabulated by Dr. Parkes, the daily secretion for his weight of 78 lb. should only have been 275 grains.

The patient continued without much change beyond a progressive increase of debility and loss of flesh, drinking enormously, and voiding corresponding quantities of urine, until July 5th, when he was suddenly seized with convulsions and insensibility. After the convulsions had ceased, he began to recover some degree of consciousness, and passed into a semi-comatose condition, which persisted for three days, and then passed away. During the period of unconsciousness the diuresis diminished notably; but it returned immediately afterwards, and the patient continued very much as he was before the seizure, for a period of ten days, when he was again taken with convulsions and insensibility, and died on the morning of July 18th.

Autopsy.—Thirty hours after death. The body was emaciated to the last degree; signs of incipient putrefaction appeared on the abdomen, the weather being warm.

Chest. The heart was healthy, but very small; the lungs were stuffed with crude tubercle throughout their upper lobes, and several small vomicæ lay scattered through them.

Abdomen. Five tuberculous ulcers were discovered in the small intestines; some of them had penetrated the mucous and muscular coats, and seemed ready to break through the peritoneum. There was no tubercular deposit in the peritoneum generally, nor any in the liver or spleen.

The *Kidneys* were voluminous, smooth, flaccid, and the two together weighed eight ounces. On section they showed no disproportion between the pyramidal and cortical portions, nor any other morbid change. Examined microscopically, the tubes and cells appeared normal.

Head. About two ounces of clear serum escaped from the arachnoid sac. The meninges were free from tubercle, and quite natural. The ventricles were greatly distended, and contained six ounces of clear serum; their parietes were macerated, and gave way with the slightest traction.

A nodule of yellow tubercle, of the size of a hazel nut, lay imbedded in the left hemisphere, in the border of the longitudinal fissure, midway between its extremities, and cropping out on the surface. Another nodule, as large as a garden-bean, was found in the posterior border of the right half of the cerebellum. An undue vascularity prevailed at a few spots of the surface of the encephalon. Apart from what has been related, the brain substance was healthy and of firm consistence. The floor of the fourth ventricle was especially examined; it was pale and natural, with no tubercular mass in its immediate vicinity.

CASE VI. (Mascarel—Gaz. d. Hôp., February 23, 1863.)—The patient was a man, aged fifty, pale and thin, without fever, but a devouring thirst, and a red tongue; appetite good, but not voracious. He drank daily from eight to ten pints of water, and voided urine proportionally. The disease had existed eight months. Seven days after entering the hospital he became feverish, at first only in the night, then continuously, with nausea, and epigastric tenderness. Thirst was intense, but there was no appetite. Not the least trace of sugar or albumen existed in the urine. The urine showed, after the fever became persistent, on cooling, a slight yellowish-white deposit, not mucous, but as if purulent. This last character was only noticed two days before death.

Autopsy.—The left kidney was more voluminous than the right, and eight to ten little abscesses, varying from the size of a pin's head to a small filbert, were found in the cortical part. The smaller abscesses contained almost concrete pus, and the larger ones fluid pus, without any tubercle. The infundibula were filled with a creamy fluid. All the abscesses were near to and reached the surface.

The right kidney was of natural size, hyperæmic, and free from disseminated abscesses, but a lactescent fluid could be squeezed from the pyramidal portions.

The brain was not examined.

Nature of Diabetes Insipidus.—A review of the *post-mortem* examinations just recorded, is sufficient to show that the initial disorder in diabetes insipidus must be looked for elsewhere than in the kidneys. The diverse organic alterations found in the kidneys by Eade, Neuffer, and Mascarel, were evidently secondary, and produced by the irritation of the frequent micturition and excessive and long-continued diuresis. Similar alterations are found in the kidneys of persons dying of long-standing saccharine diabetes. The case of Dr. Strange is certainly very puzzling: one can only conceive a teleological reason for the diuresis; namely, the absolute necessity for an immense transudation of watery fluid to make up for the imperfection of the glandular apparatus.

Nor can the disease be regarded merely as excessive thirst and a vicious habit of profuse potation. It has been almost invariably found that an enforced diminution of liquids fails to arrest the diuresis, except partially. The observations of Falck, Neuschler, and Neuffer, agree perfectly in this: that when the supply of water by the mouth is diminished, the quantity of urine notably exceeds the ingoing water, and thereby occasions dehydration of the tissues, with an intolerable aggravation of the symptoms.

It may be regarded as probable, that the *immediate* anatomical cause of polyuria is a dilatation of the renal capillaries, whereby their walls are thinned and rendered favourable to increased transudation of watery fluid from the blood. But how is this brought about? It is now generally believed that the minute blood-vessels possess in their circular and longitudinal muscular coats a provision for an active expansion as well as an active constriction of their calibre.* This provision is under the control of the sympathetic branches of nerves (nervi vasi-motores), and serves to maintain the aqueousness of the blood within certain limits of health. When the tissues and blood are overcharged with water, the renal vessels expand, and permit a copious transudation of an aqueous urine; when, on the other hand, the system is undercharged with water, they contract, and thereby restrict the urinary transudation. In diabetes insipidus this endowment seems greatly impaired; the renal capillaries appear to resemble the iris in glaucoma, which remains in a motionless, semidilated state, and neither contracts with light nor dilates with belladonna. In polyuric subjects the contractile power of the renal vessels is apparently paralysed; and the power of regulating the urinary flow consequently lost. If a healthy person imbibe an excessive amount of water, he rapidly gets rid of the overplus by a sudden and copious diuresis, and then the secretion falls quickly to its ordinary rate: but a polyuric subject, under similar conditions, shows very little *immediate* increase of urine, but a steady, persistent, though less intense, augmentation, lasting many hours, and which is not succeeded by a fall to the ordinary standard. On the other hand, if a healthy person imbibes a lessened quantity of water, the discharge of urine falls in proportion: whereas the polyuric, under the same circumstances, shows no such adaptation; he still continues to discharge an undue amount of urine, which necessitates constant imbibition of new supplies of water to prevent dehydration of the tissues.*

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* For a demonstration of the anatomical possibility of this endowment I must refer to Schiff's ingenious researches. See his Untersuchungen über die Zuckerbildung in der Leber, p. 92. Würzburg, 1859.

On this view, the primary cause of diabetes insipidus must be looked for in some other parts than the kidneys; namely, in some part of the chain of sympathetic nerves which controls the action of the contractile tissues of the renal vessels. This chain extends from the kidneys to the abdominal ganglia, thence to the spinal cord and the floor of the fourth ventricle, where the sympathetic system seems to have its centre. From above, this centre receives impressions from the encephalon.

This theory seems conformable both to experiment and to clinical facts. Bernard found that by puncturing a certain spot in the floor of the fourth ventricle, an augmented secretion could be produced of a watery urine, containing neither sugar nor albumen. A large proportion of the cases of diabetes insipidus followed injuries to the nervous centres, or were evidently dependent on some derangement of the nervous system. In the case examined by myself, palpable disease of the brain was found after death, while the kidneys were healthy. In other cases, it is probable that the sympathetic in the abdomen was the point originally injured. Among such may be classed those arising from drinking cold fluids while heated, and perhaps also those following alcoholic excesses. Another feature of the disease favourable to the theory of its nervous origin, is its occasional sudden onset after events which do not directly implicate the urinary organs; and its equally sudden subsidence when a strong impression is made on the system by an intercurrent inflammation. The total and unexpected disappearance of the disease, after continuing many months or years, is more in accordance with the habit of neuroses or nervous diseases than of any other class of maladies.*

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* See Anderzohn's Inaug. Dissertation.

The *diagnosis* of diabetes insipidus lies on the surface. A permanent increase of the urine, without sugar or albumen, suffices at once to define and to identify it. But it is evident from the facts and considerations before adduced, that to gain a useful clue for treatment, we must attain to more precise notions as to the part originally affected—whether brain, or cord, or abdominal ganglia, and also as to the nature of the lesion in the affected part.

The *prognosis* is less serious than in saccharine diabetes; nevertheless, insipid diabetes is a very unmanageable complaint; it generally resists treatment, and not unfrequently runs a fatal course. The gravity of the prognosis in a particular case depends on the severity of the general symptoms, and on the presence or absence of complications. The cases which affect the general health the least, though mostly proving incurable, appear to be those which arise after inflammatory complaints, after mental emotion, cerebral injuries, and those which arise early in life without any known cause. On the other hand, those which depend on organic disease of the spinal centres are necessarily fatal.

Treatment.—Until we obtain a better insight into the pathology of these cases, our treatment must be necessarily empirical. Hitherto the indications pursued have been confined to efforts to subdue the more palpable symptoms—the thirst and diuresis. The means used for this purpose have been various. J. Frank considered nitrate of potash in large doses as a specific: in some of the recorded cases it proved of decided service; in others it as completely failed. Camphor and valerian were used in a number of the French cases, and sometimes with success. Trousseau speaks in high terms of valerian, and cites the authŏrity of Rayer as additional evidence of its efficacy. Trousseau gave it in large doses. In one case, which ended in complete and permanent recovery in four months, the extract was gradually pushed to the enormous dose of one ounce daily; his ordinary dose would appear to be two-and-a-half drachms a day. Rayer obtained rapid success in a boy who suffered from polyuria, with emaciation and nervous symptoms, by means of the powder of valerian.

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* It has been suggested by some late writers that inosite might be found in the urine in D. Insipidus, and hold an important place in its pathology. The recent researches of Gallois negative this conjecture: see Gallois' essay De l'inosurie. Paris, 1864.

Enforced abstinence from fluids was tried in a number of cases; and, in one, recorded by Becquerel, with good effect: but in nearly all the others it was not only unsuccessful, but was followed by decided aggravation of the general suffering, and in some cases by symptoms of threatening or actual uræmic poisoning. The fate of Dr. Strange's patient is particularly instructive on this point. In one of my own cases opium produced great diminution of the thirst and diuresis, but the patient's distress was so increased that I was compelled to suspend the use of the remedy.

Among the remedies occasionally followed by success were ergot, iron, gall-nuts, lime-water, cremor tartar, iodide of mercury, and iodide of potassium.

One of the most frequent incidents in the history of diabetes insipidus is the temporary suspension of the thirst and diuresis on the occurrence of some intercurrent febrile affection, and in two instances the suspension proved permanent. A hint for treatment may be taken from this. The application of a large blister on the nape of the neck or the epigastrium (according as the associated symptoms and the anamnesis point to the nervous or the digestive system), might in some cases have the same beneficial effect as a spontaneous inflammation. In the case, treated with opium, just alluded to, a blister to the pit of the stomach proved of more benefit than any of the numerous means previously employed.

The secondary symptoms—dryness of the skin, epigastric and lumbar pains, etc., must be treated by warm baths, alkaline tonic infusions, sedative and anodyne remedies.

APPENDIX.

Cases characterized by excessive diuresis and thirst; urine of very low specific gravity, but containing, or having contained, a trace of sugar.

Cases of this type form an intermediate group between insipid and saccharine diabetes; and their existence completes, in an exquisite manner, the correspondence between the results obtained by Bernard from artificial injuries to different parts of the floor of the fourth ventricle, and clinical observations.

Two cases of this class, following fracture of the skull, are reported by Fischer (Archives Gén., Oct. 1852). In one, the sugar amounted to 0·32 per cent. In the other, in which there was voracious appetite as well as intense thirst, there was 0·5 per cent. on the first day after the accident, and 0·6 per cent. on the third day. The floor of the fourth ventricle was examined in both instances after death, and was alleged to be healthy: in the second case (which terminated in tetanus), the whole brain and cerebellum, so far as could be made out, were uninjured.

A third case, arising spontaneously, is related by Trousseau. The disease had already existed four years without serious giving way of the health. The examination of the urine (by Bouchardat) on two occasions, at considerable intervals of time, showed a trace of sugar. The quantity of urine varied from 12 to 37 litres a day. Among the secondary symptoms were impotence, lumbar pains, and a remarkable tolerance of alcoholic drinks. This man derived considerable benefit, but was not cured, by valerian.

A fourth case is recorded in the Gaz. des Hôpitaux for 1861. A man, æt. 35, was afflicted for many years with polyuria, passing daily from 10 to 12 pints of urine, sp. gravity 1001—1007. He was the subject of chronic phthisis when in the Hôtel Dieu under Trousseau in 1861. There was then not a particle of sugar or albumen in the urine; but when he was an inmate of the Hôp. St. Antoine, in 1856, a trace of sugar was found. Acute pulmonary symptoms came on at last, with purpura. The urine rapidly diminished in quantity, and the patient sank. The autopsy was performed by Luys. The floor of the fourth ventricle was more vascular than natural; large vascular trunks mapped the surface; yellow spots were seen scattered over the upper part, near the crura cerebri. Similar patches were found below the points of origin of the radicles of the portio mollis. On section, the whole grey substance was found unusually vascular, and of a rosy hue. Microscopic examination showed that these alterations in colour were due to fatty degeneration of all the nerve cells of the corresponding regions.

A case which may be classed with these occurred to myself some years ago. A man of sixty-five was brought into the Manchester Infirmary in an apoplectic fit. He died after lying for six hours in deep coma. During this period he flooded the bed with urine. After death a large quantity of urine was withdrawn from the bladder. It had a sp. gravity of 1010, and contained a considerable quantity of sugar. A voluminous clot was found in the brain.

CHAPTER II.

DIABETES MELLITUS.

Prout—Stomach and Renal Diseases, chap. ii.

Bouchardat—Annuaire d. Therap. 1841, 159; 1846, Suppl. 162; 1848, 227; 1849, 136; 1855, 147; 1865, 291; and Clinique Europ. 1859, 217.

Garrod—Gulstonian Lects. on Diabetes. Brit. Med. Journ. 1857.

Griesinger—Studien über Diabetes. Arch. d. Phys. Heilk. 1859.

Pavy—Diabetes: its nature and treatment. Lond. 1862.

Seegen—(Etiology of Diabetes). Archiv. f. path. Anat. xxi. 218.

Fritz—Du Diabète dans ses rapports avec les maladies cérébrales, Gaz. Hebd. 1859, 264.

Fischer—Diabète consécutif aux traumatismes. Arch. Gén. 1862.

Bernard—Leçons de physiologie. Paris, 1858; and Clinique Europ. 1859, 81.

France—(Diabetic cataract). Guy's Hosp. Rep., 3rd series, vol. vi., 226.

V. Graefe—(Diabetic cataract). Archiv. f. ophthalm. 1858.

Lécorché—(Amblyopie diabétique). Gaz. Hebd. Nov. 1861.

Marchal de Calvi—Recherches sur les Accidents Diabétiques. Paris, 1864.

Roberts (W.)—On the treatment of diabetes. Brit. Med. Journ. 1860.

Gray—(Treatment). Glasgow Med. Journ. vol. iv.

Schiff (J. M.)—Untersuchungen über die Zuckerbildung in der Leber. Würzburg, 1859.

The multiplied researches of recent years on the occurrence of sugar or glucose in the urine, necessitate the adoption of some classification of cases of saccharine urine.

Cases of saccharine urine may be primarily divided into two broad classes or divisions.

One class consists of instances in which a small quantity of sugar appears in the urine for very short periods, without relevant symptoms—the circumstance being a temporary and incidental consequence of some physiological or pathological antecedent which has little or no affinity to diabetes, as clinically understood. Belonging to this class are examples of saccharine urine after the administration of chloroform, after eating an excessive quantity of saccharine and amylaceous food, and after a paroxysm of whooping-cough, asthma, or epilepsy. These may be designated as cases of *incidental glycosuria.*

In the other class of cases the glycosuria is more intense; it constitutes a permanent symptom, and persists for considerable periods of time, and is associated with a serious departure from health. To this class alone is the term *diabetes* at all applicable.

This second class again is divisible into two groups. In the first, the glycosuria is persistent and intense, and the flow of urine is greatly increased: this state of urine is associated with thirst, debility, emaciation, and a train of grave, fatally-tending symptoms, which constitute a familiar, easily recognised clinical unity. This is the classical *diabetes* of authors, and to this the name diabetes was limited, before our more refined and ready analyses disclosed the presence of sugar in the urine in a number of other and different states.

The second group embraces those less serious types in which sugar is present in the urine, sometimes abundantly, sometimes scantily, sometimes persistently, sometimes intermittently; always with a weakly condition of health, but without thirst or conspicuous emaciation, often indeed with corpulence; without any, or only slight, increase in the quantity of urine, and without that fixed tendency to death which stamps the first group—occurring also generally in advanced years, or at least beyond the time of early manhood. Some of these *milder* types of glycosuria will be separately noticed at the end of the present chapter.

ETIOLOGY OF DIABETES MELLITUS.

In the decade 1851-60, 4546 deaths from diabetes were registered in England and Wales, being an annual average of 454. Of the total number 3032 were males, and 1514 females, showing that diabetes is twice more common in men than women. Up to the age of puberty, the two sexes appear to be equally liable to diabetes; but from that period on to old age the liability of the male sex maintains an increasing ratio, as may be seen from the following table :—

TABLE *showing the number of Deaths from Diabetes, at different periods of life, in the two sexes.*[*]

PERIOD OF LIFE.	Under 5 yrs.	5—10 yrs.	10—15 yrs.	15—25 yrs.	25—35 yrs.	35—45 yrs.	45—55 yrs.	55—65 yrs.	65—75 yrs.	75 years and upwds.	All ages
Deaths in males .	28	40	97	378	468	502	550	500	364	105	3032
Deaths in females	23	42	78	220	282	261	247	191	144	26	1514
Total males and females .	51	82	175	598	750	763	797	691	508	131	4546

Diabetes prevails chiefly among young and middle-aged adults. It is rare under five years of age. The youngest example that has come under my notice was a boy of three years; but in the Registrar-General's Reports for 1851-60, ten deaths from diabetes under the age of one year are registered, and as many as thirty-two under the age of three years. The mortality from diabetes attains its maximum between the ages of twenty-five and sixty-five years, and maintains itself between these epochs with tolerable uniformity. In extreme old age deaths from diabetes are more rare, not only absolutely, but as compared to the mortality from all causes.

The development and exercise of the *sexual functions* appear to have a marked effect in increasing the liability to diabetes in both sexes: and the diminished frequency of the disease in women after the age of forty-five (as compared with men) corresponds with the earlier decline of the sexual activity in the female sex. The maximum mortality in males is between forty-five and fifty-five years; in females between twenty-five and thirty-five years.

Urban and manufacturing districts suffer more from diabetes than *rural* districts. If we take London, Lancashire, and the West Riding of Yorkshire, as representing the former, we shall find that they had a mortality from diabetes of 2·5, 3·2, and 4·1, respectively, for every 100,000 inhabitants, in the year 1860; while Wales and the South Midland Counties, which are mostly agricultural, had, in the same year, a mortality from diabetes of 1·2 and 1·5, respectively, per 100,000 inhabitants. The difference does not depend on the greater *general* mortality in urban populations. For if we take the proportion of deaths from diabetes, and compare them with the deaths from all causes, we shall find that, for every 1000 deaths from all causes, there were 1·1 deaths from diabetes in London, 1·4 in Lancashire, and 1·8 in the West Riding: whereas in Wales the proportion was only 0·6, and in the South Midland Counties, 0·8.

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[*] Constructed from the Registrar-General's Reports, for 1851—60, for England and Wales. Mean population for the decade, 19,000,000.

Hereditary influence is not a prominent predisposing cause of diabetes. There are however a number of instances on record in which the disease has run in families. Seegen mentions a brother and sister who were both diabetic. A short time ago I had under my care an uncle and niece similarly affected. I have also a note of a family of eight children, all of which became diabetic, though the parents were healthy. Sir H. Marsh* refers to a family in which diabetes could be traced to the second generation, and to another family in which it could be traced through four generations. Diabetic patients have frequently been observed to belong to families in which phthisis and epilepsy prevailed. One of my patients was one of four survivors out of a family of twenty-five; twenty of these had died, after reaching adult age, of lingering complaints with great emaciation, probably phthisis or diabetes.

EXCITING CAUSES.—The exciting cause of diabetes is often obscure. In a considerable number of cases the disease has broken out soon after exposure to *wet and cold;* in others after copious drinking of cold fluids while in a heated state. Cases arising from cold, generally present a train of neuralgic or so-called rheumatic symptoms, before the breaking out of the thirst and diuresis. Excessive use of *saccharine and amylaceous* articles of food, antecedent *acute febrile diseases*, abuse of *alcoholic* drinks, have all been noted as probable exciting causes of diabetes.

The disease is sometimes traced to a violent *mental emotion*. In one of my patients it followed on distress of mind caused by an unjust suspicion of theft; in another it followed the burning down of his place of business; in a third it was attributed to anxiety attendant on a chancery suit. Rayer mentions a case of diabetes coming on after a violent fit of anger, and Landouzy another after violent grief.[*]

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* Dublin Quarterly, 1854, p. 17, note.

Organic diseases of the brain and cord have, of late years, been shown to be occasionally the exciting cause of diabetes. These cases are of special interest as bearing on the discoveries of Bernard, Schiff, and Pavy, on the artificial production of glycosuria in animals by cutting or puncturing various parts of the nervous system. Pavy cites the following examples:—A late alderman of the city of London was seized with cerebral hemiplegia. "His urine was tested by Dr. Barlow at the period of the attack and found to be free from sugar. There had also been nothing from the symptoms and history to lead to the suspicion that sugar would be found. Shortly afterwards, however, strongly marked diabetes set in. A member of the medical profession, who was seen by Dr. Gull, was seized at the age of fifty-two with an apoplectic fit, from which he recovered, with hemiplegia, however, of the left side of the body remaining behind. Five weeks after the fit, this person who had never previously presented any symptoms of diabetes, began rapidly to emaciate, which led to an examination of the urine being made. A highly saccharine state of it was found to exist." (P. 124.)

E. Fritz has collected an interesting series of cases of diabetes associated with various other organic diseases of the brain and cord (cerebral softening, tumours of the pia mater, general paralysis, and myelitis).

Still more numerous are the cases in which diabetes has followed *external injuries* to the brain and other parts. Dr. Goolden[†] has published a series of such examples; and P. Fischer has increased the list to twenty-one cases, and ably analysed them. The injuries consisted of blows and falls on the forehead, vertex, and occiput—sometimes with and sometimes without fracture of the skull. In some instances there was temporary loss of consciousness, in others not.

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[*] It has been alleged that diabetes may follow the bites of rats and venomous serpents. An examination of the facts on which this allegation is based, shows it to be without probable foundation.

[†] Lancet, June and July, 1854, and Med. Times, Dec. 1854.

In addition to cases of violence directly affecting the brain, a large number (twenty-two cases) are cited by Fischer of blows on the face, fractures of the vertebræ, blows on the loins, the thorax, the abdomen, contusions of the kidneys and liver, violent efforts, etc., which have been followed by diabetic symptoms.

Some of these traumatic cases were examples of confirmed diabetes of the ordinary type, and ran a fatal course. Others were of much milder type, transitory in their duration, passing away on the subsidence of the cerebral symptoms. In some of them the diabetic symptoms commenced at the time of the accident, or shortly after; in others, not until some months had elapsed after the injury.

In a certain proportion of the traumatic cases (eight out of forty-three collected by Fischer) the urinary disorder consisted of simple polyuria.

It is probable that in all the traumatic cases the injury (however different its seat) implicated some part of the sympathetic nervous system, either within the cranium or spinal cord, or in its peripheral distribution.

SYMPTOMS.

The invasion of diabetes is generally insidious. The disease is seldom recognised until it has existed some weeks or months. The initial symptoms (malaise and slight emaciation) pass unnoticed, because the appetite continues good; but the patient's suspicions are at length aroused by the increasing frequency of the calls to make water, and an incommodious thirst. The disease sometimes, however, attains a high intensity in a few weeks. In one of my cases as much as fifteen pints of urine a day were secreted in the third week.

As the disease advances, it assumes its characteristic features. The thirst becomes insatiable, the appetite excessive or voracious, the skin harsh, dry, and scurfy; the patient emaciates; the countenance wears an appearance of suffering, and the features are drawn; a distressing sinking is felt at the pit of the stomach; the tongue is glazed and furrowed; a scanty, tenacious mucus gathers in the mouth, which is parched and clammy; the urine rises to eight, twelve, or more pints in the twenty-four hours; this urine is of a pale straw colour; its density varies from 1035 to 1045 or higher; and it contains a large proportion of sugar; the virile powers fail; and the mental faculties lose their wonted vigour.

If the malady proceeds unchecked, these symptoms increase in intensity. The emaciation and loss of strength attain an extreme degree; pulmonary symptoms resembling those of phthisis often make their appearance, and advance with alarming rapidity; or colliquative diarrhœa sets in; hectic fever is established; the urine now diminishes in quantity, perhaps loses its sugar and becomes albuminous; the legs become œdematous; and the unfortunate sufferer is at length released, often very suddenly, either by sheer exhaustion, or he is carried off by one of the numerous complications of the disease.

Some of these symptoms require a more detailed consideration.

The urine.—The essential features of the urine in diabetes are its excessive quantity and the presence of sugar. The proportion of the latter varies from 8 to 12 per cent. It is chemically identical with grape-sugar, or glucose. The quantity excreted daily ranges from 15 to 25 ounces; but it may amount to two pounds or more, or fall to an ounce or less. The proportion of sugar is always increased after food, and diminished after fasting. After the use of starchy or saccharine substances, the increase is much greater than after animal food. In many of the milder cases, and probably in the earliest stages of all, the urine becomes free from sugar when starch and sugar are entirely withdrawn from the diet; but in confirmed cases the urine still continues saccharine—though in greatly diminished intensity—when the diet is purely animal, and even when no food at all is taken. In this last case the sources of the sugar are necessarily the tissues of the body.

The density of diabetic urine usually fluctuates a few degrees above or below 1040; it may rise to 1055 or 1060, or sink to 1015.

Intercurrent inflammatory or febrile attacks cause the sugar to diminish, or even to temporarily disappear. Toward the approach of death a similar diminution is observed.

The quantity of the urine oscillates usually between 8 and 15 pints daily; but it has been known to exceed 32 pints. When the diet is restricted to animal food, the urine is generally reduced to four or five pints a day. The quantity of the urine is about equal to the liquids imbibed. The opinions formerly held, that the urine exceeded the ingested liquids, and that the body absorbed water through the lungs and skin, or generated it *de novo* from the elements of the food and tissues, are quite unsupported by more rigid observations of recent date.

When the flow is considerable the urine has a very pale straw tint, and a peculiarly bright aspect. It speedily becomes opalescent when exposed to the warm air, and in a few hours ferments, with abundant disengagement of gas and production of sporules and filaments of the yeast plant. These latter form a white flour-like deposit in diabetic urine after it has been kept awhile.

When the flow does not exceed four or five pints, the colour and general appearance of the urine are natural.

With regard to the ordinary constituents of the urine, no particular alteration takes place in their rate of excretion beyond a diminution of their proportion to the water, and (generally) some absolute increase of their quantity. Very contradictory statements have appeared on this point: but the more trustworthy observations appear to support the above conclusion, especially with respect to urea. Uric acid is often difficult to detect, owing to the immense proportion of water; but it is not really absent, as has been alleged; and when the volume of the urine is reduced to four or five pints a day, uric acid frequently forms an abundant deposit of large dark-red crystals. More rarely I have observed oxalate of lime; and in one instance a persistent deposit of crystallized phosphate of lime.

The presence of a small quantity of albumen, and even of blood, is not uncommon in advanced cases, and constitutes an untoward sign. In a gouty old gentleman who was passing four pints of a moderately saccharine urine a day, I detected (in addition to a little albumen) transparent fibrinous casts of the uriniferous tubes.

Thirst is one of the earliest and most persistent symptoms of diabetes, and has often led to its detection. Diabetic patients will generally drink from 8 to 12 pints a day; but sometimes they imbibe as much as 25 and 35 pints a day. Nevertheless, this enormous potation does not suffice to quench the intolerable thirst—nay, it seems even to intensify it. A perpetual painful dryness of the mouth and fauces remains in spite of a deluge of water. Patients have even been known to drink their own urine to allay their craving for fluids.

The immediate cause of the thirst is, probably, the existence of sugar in the blood. This crystalline substance, like any other crystalloid, creates a demand for water to effect its dissolution and elimination from the body. On the other hand the consumption of large quantities of water seems to aggravate the disease, by accelerating the disintegration of the tissues.

Inordinate *appetite* is not nearly so constant a symptom as excessive thirst; and in the course of the complaint there is not unfrequently complete anorexia. Toward the fatal termination a loathing for food of every sort often prevails, and is accompanied by rapid sinking of the powers of life.

Emaciation is generally a prominent symptom; and the degree of it is proportional to the intensity and duration of the disease. The disappearance of fat is probably not without direct connection with the unnatural transformation of the amyloid substance of the liver into sugar; as it seems highly probable that the normal destination of this is, partly at least, to nourish the adipose tissues of the body. Emaciation is, however, not an invariable concomitant of diabetes. One of my patients weighed over 15 stone, though he had been voiding daily 12 pints of a highly saccharine urine for some months. One of Prout's diabetic patients weighed 23 stone! This obese class of cases are markedly less severe, and of more hopeful prognosis than the generality.

The emaciation is not confined to the fatty tissues: the muscles become atrophied, and even the heart itself. The enormous flow of fluid through the kidneys explains to some extent the excessive emaciation of diabetic patients; for it has been shown by Genth, Böcker, and Mosler, that simple transudation of water through the body increases the disintegration of the tissues, and induces rapid loss of weight, unless the deficiency be made up by increased supplies of food. In diabetes, notwithstanding the enormous amount of aliments ingested, the defective state of the digestive organs prevents the possibility of suitable compensation by increasing the supply from without. In agreement with this view, it is found that when the flow of urine in diabetic patients is brought down by appropriate treatment to three or four pints a day, there is usually no further loss of weight, or even the patient recovers some of what he has lost, though the urine still continue saccharine.

Dryness of the *skin* is a usual and very unpleasant symptom of diabetes, and its intensity is proportional to the diuresis. Some diabetic patients, however, sweat freely throughout their complaint ; others only begin to sweat on the advent of hectic fever.

The prevalence of *boils* is a curious occasional coincidence of saccharine urine. In a gentleman recently under my care, successive crops of boils were the earliest symptom of the disease. Sometimes they constitute veritable furunculi, and as many as twenty-two have been counted at the same time on a diabetic patient. They may even be the immediate cause of death. Other cutaneous affections are sometimes seen, but less frequently than boils, namely, psoriasis and impetigo.

An erythematous and excoriated condition of the urethral orifice (due to the irritation of the saccharine urine) is occasionally a source of great discomfort ; and in the female, heat and itching about the vulva is a common and distressing symptom.

Dr. Garrod states that *œdema of the legs* is a constant feature in diabetes. It certainly is very common ; and the flat surfaces of the tibiæ can nearly always be made to pit on firm pressure, even when no fullness exists about the ankles. I am satisfied, however, that this pitting, when very slight, is not due to œdema, but rather to the soft atonic state of the subcutaneous tissues.

Ascites and *œdema of the arms and hands* occurred in one of my cases. Ascites is also mentioned in one case by Fischer, where the disease was complicated with cataract.

The dryness of the *mouth* usually corresponds to that of the general surface. The tongue is commonly red, preternaturally clean, cracked, and denuded of epithelium. In the less severe cases, or when amelioration has been brought about by treatment, the tongue is moist, and coated with a thin yellowish-white fur. In the majority of diabetic patients the *teeth* are gradually destroyed by caries; and the gums become spongy, swollen, loosened from the teeth, and liable to bleed. The destruction of the teeth is attributed by Falck to the excessive acidity of the saliva, due to the generation of lactic acid from the sugar present in the secretions of the mouth. Sometimes, however, the teeth are preserved perfectly, in persons who have been diabetic for many years.

The *digestive organs* rarely bear the unnatural strain put upon them by the excessive feeding of diabetic patients, without at length resenting the ill-usage. Epigastric pains and a sense of sinking at the scrobiculus cordis, flatulence, and occasional vomiting, are the most common symptoms. As a rule, the bowels are constipated, and require artificial aid to promote their action. The fæces are generally pale. In advanced cases diarrhœa not unfrequently occurs, sometimes of a dysenteric character. This is a formidable and generally speedily fatal complication.

The *mental state* suffers a marked alteration in confirmed cases; but the degree and type of it varies a good deal. The change most commonly observed is a heaviness and apathy, a disinclination to mental and bodily exertion, sometimes a positive drowsiness. The natural firmness of the character gives place to a deplorable pusillanimity and a want of moral sense, which are foreign to the individual in a state of health. Persons who previously had been above every equivocation or concealment, resort to petty cunning and positive untruthfulness to deceive their medical attendant as to their food and drink. Nevertheless, the intelligence itself is not troubled, and continues clear to the end.

The *sexual functions* undergo a notable declension of vigour in advanced cases; and there is frequently actual impotence from failure of the power of erection. This defect, however, is not a permanent one; and if amelioration take place the virile powers return early. Exceptions to this rule also occur. Dr. Prout mentions an instance of a confirmed diabetic, who married and had two children, though the saccharine condition of the urine still persisted.

The *blood*, as might have been expected, is unnaturally charged with sugar in diabetes. It has been found in the proportion of from 0·3 to 0·5 per cent. From the blood, sugar finds its way into all the tissues and fluids of the body. Sugar has been found in the fæces, the sweat, the humours of the eye, the tears, the saliva, and the gastric juice.

COURSE, DURATION, TERMINATION.

Diabetes is an essentially chronic disease; its course is measured by months and years. The ordinary duration of diabetes is from one to three years. Sometimes the disease runs a rapid course and terminates in a few months or weeks. The most rapid example which I have seen was a child of three years, who died in three weeks. Becquerel mentions the case of a boy of nine years who died in six days. On the other hand, cases sometimes run on for six, eight, or ten years. The following table shows the duration of diabetes in 100 fatal cases, collected by Griesinger:—

Under 3 months	1 case.
Between 3 and 6 months	2 cases.
,, 6 ,, 12 ,,	13 ,,
,, 1 ,, 2 years	39 ,,
,, 2 ,, 3 ,,	20 ,,
,, 3 ,, 4 ,,	7 ,,
,, 4 ,, 5 ,,	2 ,,
,, 5 ,, 6 ,,	1 ,,
,, 6 ,, 7 ,,	2 ,,
,, 7 ,, 8 ,,	1 ,,
Undetermined	12 ,,

The progress of diabetes is usually equable and continuous; but cases are met with, not very unfrequently, in which the symptoms intermit—the saccharine state of the urine ceasing and recurring at intervals. Dr. Bence Jones* has published an account of several such cases in old persons; and I have encountered three similar ones in my own practice. These will be again noticed among the milder types of the disease. Girard records an example of intermittent diabetes in a girl of eighteen.*

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* Med. Chir. Trans., vol. xxxvi.

If diabetes does not terminate through one of its complications, the patient becomes gradually drowsy, and finally dies comatose—sometimes with the occurrence of convulsions. Not unfrequently death is at the last sudden. One of my patients fell back dead while eating his dinner.

The symptoms sometimes begin suddenly, and not insidiously. Not unfrequently too, the symptoms are much more violent in the first few months than at a later period, when the disease has become confirmed. When diabetes has already existed two or three years the thirst and voracity rarely maintain their primitive intensity. This change from a more acute to a more chronic state must not, of course, be mistaken for a real improvement.

COMPLICATIONS.

The complications of diabetes assume a prominent place in its history, inasmuch as the disease only exceptionally proves fatal solely through its own intensity. More frequently an intercurrent disorder supervenes, which carries off the patient. The most common and formidable complication is pulmonary tubercle, which affects nearly one half of the cases protracted to the third year. The pulmonary disease runs the course of rapid phthisis. A low and fatal type of inflammation is also liable to arise in the lungs, pleura, or peritoneum. In every tissue of the body there exists a tendency to asthenic inflammation, apt to run on to abscess, diffuse suppuration, sloughing, phagedænic ulceration, or gangrene.

The occurrence of boils and carbuncles in diabetic patients has long been known. The statement of Prout that sugar is always present in the urine of patients suffering from boils, is certainly incorrect. Dr. Goolden mentions the case of a medical man, long diabetic, who had an enormous anthrax on the nucha, which compromised his life. He recovered, however, from the anthrax, and with its disappearance the sugar departed from the urine. P. Frank cites an almost similar case. Phillipeaux and Vulpian (Gaz. Hebd. Dec. 6, 1861) relate an example of anthrax in a hemiplegic patient who was not previously diabetic. During the suppuration of the anthrax the urine became strongly saccharine; but ceased to be so when the anthrax cicatrized.

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* Union Med. 1855.

Gangrene of the lower extremities, resembling gangræna senilis, has been several times observed in diabetes. Sir H. Marsh[*] mentions the case of a lady, about seventy years of age, suffering from diabetes of long standing, who was carried off suddenly with gangrene of the foot and leg. On examination an obstruction was found in the iliac artery. Dr. Colles (quoted by Marsh) had seen two similar cases of obstructed arteries, and fatal gangrene in diabetes. In 1845, Carmichael presented to the Pathological Society of Dublin two cases of senile gangrene of the lower limbs in diabetic patients.[†] Marchal de Calvi has drawn attention to the same subject more recently, and published four new cases. Additional cases have also been brought forward by Hodgkin, Landouzy, Champouillon, Billiard,[‡] and others.

Defects of sight in connection with diabetes have of late years attracted a good deal of attention. They consist either in an enfeeblement of vision (amblyopia), or cataract.

Amblyopia occurs, according to Bouchardat, in about a fifth of the cases of diabetes. Generally speaking, it is slight in degree and temporary, or recurrent. In one of Griesinger's patients the amblyopia ceased when a flesh diet was used, but it was succeeded, shortly after, by cataract. Permanent amblyopia is less frequent; it occurs only in advanced cases, and is a sign of approaching fatal termination. The dimness of sight steadily increases, and at length ends in total blindness. It seems to be owing to atrophy of the retina.[§]

Diabetic *cataract* was first noticed in this country by Mr. France, and Lécorché has given an excellent resumé of what was known on the subject up to 1861. It occurs generally in inveterate cases of long standing. It is an unfavourable sign; and death follows its appearance usually in a few months; but sometimes patients with diabetic cataract survive for years. The frequency of cataract in diabetes, has been estimated very differently by different authors. Griesinger observed cataract in three out of his own seven cases. V. Graefe estimates the proportion as one in five; Bouchardat as one in thirty-eight. Garrod had not encountered cataract in any of the large number of cases of diabetes which he had seen. Out of forty-five cases which I have treated, only one had cataract. Of 225 cases collected by Griesinger, cataract occurred in twenty.

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[*] Dublin Quarterly, 1854.

[†] Med. Gaz. 1846, p. 110.

[‡] See Charcot, Gaz. Hebd. Aug. 1861; and Dr. Hodgkin, Assoc. Med. Journ. 1854. The whole subject is treated exhaustively by Marchal de Calvi in his recent work, cited at the head of this chapter.

[§] For further information on this subject I must refer to Lécorché's articles, "De l'amblyopie diabétique." Gaz. Hebdom., Nov. 1861.

Diabetic cataract comes on, sometimes without previous defect of vision, sometimes after one or more attacks of temporary amblyopia; sometimes it complicates permanent amblyopia. It generally arises after the diabetic state has lasted eighteen months or two years; but it has been known to appear in six months. Its appearance may coincide with aggravation, amelioration or stationary condition of the proper diabetic symptoms. Its course is rapid; the two eyes may become completely cataractous in a few days; sometimes it is developed more slowly. It begins in one eye—generally the right—but soon involves the two. In the case which I observed, a woman, twenty-four years of age, had been diabetic for two years and a-half. Three months previously the left eye became suddenly cataractous: in less than a week the opacity had reached its maximum. The right eye was still clear and vision perfect.

Cataract in diabetes is nearly always of the soft kind; but examples of hard diabetic cataracts have been met with by V. Graefe, Guersant, and Sir W. Wilde.

It has been conceived by Weir Mitchell* and Dr. Richardson † that diabetic cataract is produced by physical imbibition into the lens of the saccharine matter of the aqueous humour of the eye. This opinion is based on the temporary opacity produced in the crystalline lens of the frog, when the animal is immersed in a saccharine solution, or when a similar solution is injected into the cellular tissue. It is very doubtful however whether the two conditions are really pathologically analogous. Lécorché failed to produce opacity in the lens of rabbits by injecting syrup into the eye. Artificial cataract in the frog speedily disappears when the animal is removed from the saccharine solution: but diabetic cataract is permanent, and does not disappear when amelioration of the symptoms takes place. How, on the imbibition theory, can the cases be explained, in which (as in the instance which occurred to myself) one lens has been completely opaque for months while its fellow still remains perfectly transparent? How also should its occurrence be (as a rule) so long delayed, and arise so suddenly, without any corresponding increase in the quantity of sugar in the urine? It seems more probable that diabetic cataract is one of the many degenerations of a low inflammatory type so common in confirmed diabetes.

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* American Journ. of Med. Sc., Jan. 1860.

† Brown-Séquard's Journ. de Physiologie, July, 1860.

Hepp failed to find sugar in a cataractous lens removed from a diabetic patient. Fischer obtained a similarly negative result in another case. But Stœber found sugar in a lens examined by him.*

Operations for diabetic cataract generally fail, from uncontrollable suppuration of the eyeball. Sometimes, however, the operation succeeds; and if the primary complaint be stationary and quite uncomplicated, operation may be recommended as a possible solace to the remainder of life.

MORBID ANATOMY.

Although diabetes is a frequently fatal disorder, necrology has hitherto thrown little light on its seat and nature.

The more palpable anatomical changes which have been found, are manifestly not due to the disease itself, but to some of its numerous complications. Physiological data would lead us to look for the primary seat of diabetes in the liver, or in some part of the sympathetic nervous system connected with the liver. This is an extensive and difficult field for investigation; hence probably the slender results hitherto obtained from its exploration.

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* Annales d'Oculistiques, xlviii., p. 192.

The liver has certainly not yet given up its secret, if it have any. The accounts of its appearance, after death from diabetes, are contradictory. Its size is usually normal; sometimes it is a little enlarged, sometimes a little atrophied. In some cases it is congested, in others the reverse. Occasionally, it contains a good deal of sugar—more frequently, none at all. Dr. Wilks believes that the diabetic liver presents differences to the eye which enable it to be distinguished from others: it is firm, tough, homogeneous or uniform in appearance, and dark in colour.* But other descriptions are quite at variance with this. Griesinger found the liver granular and easily torn in one case; in four others the livers were perfectly normal.

Microscopical investigations have been equally unsatisfactory. Förster and Griesinger found the liver-cells natural. Beale† and Frerichs‡ remarked a diminution or absence of fat. Pavy found the fat undiminished. Stockvis found the fat undiminished, but an unusual proliferation of the hepatic cells.

Of the bile, Dr. Pavy remarks, that in nearly all the cases in which he has specially examined it, it has presented a striking appearance, resembling a rhubarb mixture, and depositing a copious sediment consisting of columnar epithelium, and yellow, amorphous, granular-looking matter.

Prout says that he frequently observed a gorged condition of the veins terminating in the portal system.

The floor of the fourth ventricle has been examined in several recent instances. Sometimes it has been found quite natural; in other cases it was found the seat of serious pathological changes. In 1860, Luys brought before the Société de Biologie an example in which this spot was softened, highly vascular and of an unnatural brown colour. The nerve-cells were found degenerated and full of yellowish granules.§ Monneret followed up this observation with another in which similar changes were encountered in an earlier stage.* Tardieu records a case of diabetes in which there existed slight paralysis of the left side for three months: the diabetes persisted until death two years afterwards from phthisis: the medulla oblongata was found congested and of a dark grey colour.† Dr. Richardson relates a case of diabetes in which convulsions and symptoms of meningitis occurred during life: after death an ossific growth was found pressing upon the pons Varolii, and an abscess in the posterior cerebral lobes.‡

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* Pavy on Diabetes, p. 117.
† Med. Chir. Rev. 1853, p. 226.
‡ Cited by Griesinger, loc. cit., p. 34.
§ Bulletin de la Soc. de Biol., 1860.

It is clear that these observations are an insufficient basis for any theory of diabetes.

Of the secondary lesions or complications, those found in the lungs are the most common. Out of sixty-four autopsies collated by Griesinger, tubercle was found in the lungs in thirty-one, or nearly one half. Pavy and Wilks believe that the pulmonary mischief is not always genuine phthisis, even when it runs a closely similar course, but consists in a chronic inflammation leading to the breaking down of the lung tissue and the formation of cavities.

Pneumonic consolidation and gangrene of the lungs have likewise been not unfrequently found. Sometimes (not always) gangrene of the lungs in diabetes is not accompanied by the characteristic fœtor of that complaint.

In long standing cases, the kidneys are not unfrequently found seriously altered. Out of Griesinger's sixty-four autopsies renal alterations were found in thirty-two. In seventeen instances there was degeneration resembling some forms of Bright's disease, mostly with fatty degeneration of the renal epithelium. Granular atrophy of the kidneys was never found; but cysts, cicatricial spots, adhesions of the tunica propria and pyelitis. In five cases the kidneys were markedly hyperæmic, and in seven considerably hypertrophied. Prout states that the kidneys of persons dying of diabetes assume frequently a peculiar deep orange tint on exposure to the air.

The stomach is commonly found distended, and the mucous membrane thickened and softened.

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* Gaz. d. Hôp., Jan. 11, 1862.
† Med. Times and Gaz., Feb. 1862.
‡ Ib., March and May, 1862.

PHYSIOLOGICAL AND THEORETICAL CONSIDERATIONS RELATING TO DIABETES.

Much light has recently been thrown on the pathology of diabetes by the observations of Bernard and others on the presence of an amyloid substance in the liver, and the possibility of inducing glycosuria in animals by artificial means. A resumé of the present state of these questions is absolutely necessary to the comprehension of any theoretical views of diabetes.

Bernard discovered the fundamental fact, that the liver of all healthy animals contains a large quantity of a substance resembling starch or dextrine. When the liver of an animal newly killed is abandoned to itself in a warm place, it speedily becomes charged with sugar by the conversion of a portion of this substance into glucose; and when the sugar so produced is washed completely out by a stream of water, the organ abandoned to itself, as before, becomes, again in twenty-four hours, abundantly charged with sugar. This conversion goes on until all the amyloid substance is changed into sugar. The transformation here witnessed takes place by the action of a peculiar ferment which circulates in the blood.

I have obtained the amyloid substance of the liver (which has received the various names of *animal* or *hepatic dextrine* (or *starch*), *hepatine* and *zooamylum*) in the greatest purity and with the greatest ease from the liver of the oyster. The large fawn-coloured mass, which constitutes the delicacy of this mollusk, should be cut out, and plunged for a few minutes into boiling water. The hardened mass is then pounded in a mortar, mixed with a small quantity of water, and boiled so as to form a decoction. This is subsequently filtered and poured into five times its bulk of strong alcohol or glacial acetic acid. An abundant precipitation of snow-white flakes is produced. This is the amyloid substance. It is collected on a filter, washed with alcohol and dried.

When pure, hepatic dextrine is a white, tasteless, inodorous body. It dissolves freely in water, forming an opalescent solution like skimmed milk. It contains no nitrogen; its formula is $C_{12}H_{12}O_{12}$. With iodine it behaves like vegetable dextrine, yielding a deep wine-red coloration. It does not reduce the salts of copper nor ferment with yeast; but (like starch and dextrine) it is speedily converted into glucose by the contact of warm saliva, pancreatic juice, or diastase. It is similarly converted by the contact of fresh blood, which has no effect on starch and dextrine. This last property is its peculiar characteristic.

Schiff has satisfied himself, by a most ingenious set of experiments, that he has detected the exact situation and physical condition of the amyloid substance in the liver. He found it, not infiltrated or dissolved in the hepatic tissue, but collected into separate vesicles or granules precisely as occurs with starch in the vegetable kingdom. In the frog, he found under a magnifying power of 600 diameters, that the liver-cells, in addition to one or two round nuclei and twelve or twenty fat globules, contained an immense number of minute pale vesicles varying in size from $\frac{1}{1800}$ to $\frac{1}{1000}$ of a line. Within these vesicles the amyloid substance is accumulated. The outer membrane (as in the vegetable starch-granule) contains nitrogen. Nasse * has subsequently detected similar amyloid vesicles in the liver-cells of warm-blooded animals. They differ from most of their vegetable homologues in not possessing concentric markings, and in not yielding a blue coloration with iodine. I have endeavoured to verify these observations in the liver of the frog and the oyster, but without success.

Animal dextrine is always present in the livers of all the healthy animals hitherto examined, whether living on vegetable or animal food, or fasting. But under a variety of diseased and unnatural conditions it quickly disappears. The circumstances preceding death from disease, are such, that the liver scarcely ever contains a trace of amyloid substance when examined *post mortem*.

The *peculiar ferment* of the liver-dextrine exists in the blood, but has not yet been isolated. It is not liable to disappear under those conditions of disease which cause the hepatic dextrine to vanish so quickly. Nevertheless it is sometimes absent. Schiff made the curious discovery that this ferment totally disappears from frogs during the second half of the winter and the early spring months. This occurs as a regular event in the annual changes which these batrachians undergo. During this interval the liver is as full as usual of amyloid substance, but no spontaneous production of sugar occurs when the organ is abandoned to itself in a warm place, and artificial glycosuria cannot be engendered in such animals. When, however, the blood of another animal, which is not in this peculiar condition, is injected into the bodies of winter frogs or applied to their livers, the usual production of sugar takes place rapidly. This absence of a ferment has not been noted, as a regular occurrence, in any warm-blooded animals; but Dr. Pavy states that he once encountered a healthy rabbit in this condition.

Great divergence of opinion prevails as to the destiny of the liver-dextrine during healthy life. Most physiologists as yet adhere to the view of Bernard, and believe that a continual conversion of this substance into sugar is going on in the liver, and that a quantity of sugar is being constantly poured into the hepatic veins and carried off into the circulation. Dr. Pavy, on the other hand, contends that there is no conversion of hepatic dextrine into sugar during healthy life, nor any continual stream of sugar flowing into the circulation; and that when such conversion does take place it is an abnormal or diseased occurrence, or due to *post-mortem* changes.

In cold-blooded animals the view of Pavy is unquestionably the correct one. I have repeatedly tested the point in frogs and oysters, and have never succeeded in detecting a trace of sugar in the liver, if the organ was examined before the possibility of any *post-mortem* changes. In the case of the oyster the experiment is a very easy one. A fresh oyster is cut in half with a pair of scissors, in such a way that one half shall drop into a capsule of boiling water: the other half is laid aside in a warm place. The latter very speedily becomes abundantly saccharine; but in the former the ferment has been rendered inert by the heat, and not a particle of sugar can be detected in it, even after being long kept in a warm place. The conditions in the oyster's liver are precisely similar (as far as is known) to those in the liver of a warm-blooded animal. An abundance of amyloid matter and the ferment co-exist side by side in the organ, and yet no re-action takes place between them, and no sugar is produced, so long as the healthy state is maintained.

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* Archiv des Vereins für gemeinschaftl. Arbeiten, IV. I., p. 97.

A similar experiment is more difficult in warm-blooded animals, because it is impossible to proceed with the same celerity; yet in Dr. Pavy's hands the results obtained were fully confirmatory of his doctrine. Bernard's glycogenic theory rests chiefly on the fact that in newly-killed animals the blood of the hepatic veins has been found sensibly richer in sugar than that of the body generally. Pavy attributes this result to rapid changes which take place during the performance of the experiment. He has varied the proceeding in such a manner as to avoid these disturbances. He catheterized the right heart by introducing a tube along the jugular vein. In this way, if the animal remained quiescent, the blood of the hepatic veins was obtained in its normal state. Hepatic blood so obtained, was found to contain only those minute traces of sugar which exist in every part of the circulation.

Dr. Robert McConnell,* in an admirable series of researches, has repeated and varied the experiments of Pavy, and obtained results which do not seem to admit a possibility of doubt, that amyloid substance is *not* converted into sugar in the liver during healthy life. In his recent memoir on the functions of the liver†, McConnell brings forward some facts and considerations of great weight in support of his view, that the real destiny of the liver-dextrine is to unite with nitrogen (set free by the disassimilation of fibrine and a portion of the albumen of the portal blood) so as to constitute a new protein compound resembling casein, which is being constantly poured into the circulation through the hepatic veins.

Dr. Pavy appears to insist too absolutely on the absence of any unimpeachable evidence of the disappearance of sugar introduced into the blood, except by its removal through the kidneys. It has been fully made out that sugar and dextrine may be injected continuously into the blood in certain small quantities—that is, so much that the per centage of them in the blood shall never rise beyond 0·2 or 0·3—without producing saccharine urine.‡ What becomes of sugar so introduced is doubtful. It may not be oxidized, as has usually been believed, into carbonic acid and water; perhaps it is transformed into amyloid substance and lodged in the liver. That it disappears, somehow, without escaping with the urine, cannot admit of doubt. Some experiments of Schiff appear to bear decisively on this point. He induced artificial diabetes in frogs by puncturing the spinal cord; he then ligatured portions of the liver, so that the discharge of sugar into the circulation was diminished in proportion to the size of the piece of liver included in the ligature. When a piece equal to about a fifth part of the organ was included in the ligature, sugar was still poured into the circulation, *but not in sufficient quantity to produce glycosuria.*

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* See Proceedings of the Royal Irish Academy, Feb. 13, 1860. The only experiments unfavourable to Pavy's view, as yet published, are those of Harley (Proceedings of the Royal Society, vol. x. p. 289). These are evidently not of sufficient weight to be placed in competition with the laborious and numerous experiments of Pavy and McConnell.

† Observations on the Functions of the Liver, by R. McConnell, M.D. Dublin and London, 1865.

‡ Schiff, l. c. p. 134.

Artificial glycosuria and diabetes. We are led to believe then, on the evidence above adduced, that although the amyloid matter and its ferment must be in close proximity in the hepatic tissue, they do not come into actual contact and react upon each other during healthy life; but they may be brought into conjunction under a variety of unnatural conditions induced by disease or injury; and physiologists are able to bring about these abnormal conditions at will, and cause sugar to appear in the urine.

Artificial glycosuria may be produced in various ways, namely, by cutting or puncturing diverse parts of the nervous centres and certain organic nerves; by impeding respiration; putting animals under the influence of anæsthetics and tetanizing substances; injecting acid substances into the portal veins; and thrusting needles into the liver.

Most, if not all, of these injuries, different as they appear, act finally in the same manner, and cause dilatation of the hepatic blood-vessels, and consequent hyperæmia of the organ. This dilatation may (conceivably) be brought about in two ways: either by an increased action of the longitudinal muscular fibres (dilating muscles) of the small vessels*—this would be an active congestion—or, by a paralysis of the circular fibres, whereby the vessels would give way and expand before the propulsive action of the heart.

The contractile tissue of the hepatic vessels, like that of the vascular system generally, is under the control of a distinct nerve-arrangement, with a local centre in its neighbourhood (probably the cœliac ganglion) and upward prolongations by the sympathetic and the spinal cord into the cerebral centres. The separate threads of this communication are, in the lower parts of their course, placed widely apart; but they approach in the spinal cord, and in the floor of the fourth ventricle are collected into a close bundle before their final dispersion into the cerebral hemispheres.

An irritation applied to any part of this nervous communication may cause temporary glycosuria; and in the floor of the fourth ventricle, even the puncture of a needle, if it be made exactly at the right spot (midway between the origins of the auditory nerves), is sufficient. The difficulty of exactly hitting this spot renders the operation somewhat uncertain, except on condition of injuring the surrounding parts extensively; and Schiff found it preferable to pass in a needle and destroy the whole thickness of the cord at the point of origin of the brachial nerves. This operation never fails to produce *temporary* glycosuria. In warm-blooded animals the urine continues saccharine for a few hours; in frogs about four days. Schiff gives good reasons to consider that glycosuria so produced is caused by an active congestion of the liver.

The *permanent* diabetes, with which practitioners are familiar in the human subject, appears, on the contrary, to be paralytic in its nature, and to be due to a passive congestion of the liver from loss of contractility in the circular fibres of the hepatic vessels. Schiff succeeded in inducing, in rats, a permanent diabetes which may be looked on as the true counterpart of the spontaneous disease in man. This was accomplished by operating on the spinal cord at a lower point. He passed a strong needle into the spinal cord (with the least possible injury to the surrounding parts) and destroyed it, opposite the second dorsal vertebra. Rats operated on in this way, lived, provided their temperature was artificially sustained, for seventeen and even twenty days, and continued diabetic to the end. Rabbits sometimes outlived this operation nine days, and continued diabetic to the last day. Animals higher in the scale than rodents do not survive this operation.

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* See Schiff, loc. cit. p. 92.

Temporary glycosuria has also been induced by impeding respiration (Pavy); by poisoning with strychnia and woorali; by thrusting needles into the liver (Schiff); by chloroform and ether inhalations in warm-blooded animals; in frogs, by tying the afferent veins of the kidneys so as to increase the flow of blood through the liver (Schiff); by injecting acids into the veins.

It should also be mentioned that the introduction of large quantities of sugar and starch by the digestive organs occasions glycosuria,—showing that the assimilating power of the liver over these aliments is not unlimited. Inuline (which replaces starch in the compositæ) induces slight glycosuria, even when partaken of in comparatively small quantity (Schiff).

Bearing these physiological data in mind, we shall not find any difficulty in explaining the circumstances under which temporary glycosuria occurs in the human subject in connection with various injuries and diseases; and we obtain some dim insight into the true pathology of clinical diabetes.

It must be remembered, in searching for sugar in the urine of persons who present the alleged conditions of glycosuria, that the search will be in vain if there be great disturbance of the general system, and especially if there be fever: for the amyloid substance speedily disappears from the liver under these circumstances, and consequently no sugar can appear in the urine, however perfectly all the other conditions for its occurrence exist. This is doubtless the reason of the many contradictory results of bed-side observations on the occurrence of saccharine urine. I have repeatedly examined the urine of patients with obstruction in the chest (emphysema, &c.) in whom there existed great hyperæmia of the liver, without finding sugar: but it nearly always happens in such cases that the general well-being of the patient is deeply affected, or, that there is positive pyrexia. We ought to find saccharine urine most constantly, after paroxysms of whooping cough and spasmodic asthma, in the early stages of tetanus, after chloroform-inhalation by healthy persons, soon after injuries involving the nervous centres or the liver, and in apoplectic seizures.

Although we appear to be approaching an exact knowledge of the pathogenetic elements of glycosuria, it is yet manifestly impossible, in the present state of science, to frame a comprehensive and clear theory of diabetes. It would seem highly probable that diabetes consists proximately in some disturbance of the destiny and function of the amyloid substance of the liver. But this disturbance may be due originally to disease far away from the liver itself, in some part of the sympathetic chain which controls this function. Occasionally, as in traumatic cases, it is possible to place the finger on the primary lesion; but in the immense majority of cases we are left in a sea of conjecture. Further researches, conducted in the light of past and future physiological discoveries, can alone reduce these conjectures to order and certainty.

DIAGNOSIS AND PROGNOSIS.

The *diagnosis* of diabetes is generally a very simple matter, when attention is once directed to the urine—the existence of sugar in the urine, and diuresis, being the only points to be ascertained. The means of detecting sugar and of estimating its quantity have already been fully considered. (See p. 128.)

Care must be taken, however, not to conclude too rashly that this formidable disease exists, from the mere finding of a little sugar in the urine. It has just been shown that the urine becomes temporarily saccharine under certain conditions quite apart from genuine diabetes. Before the existence of diabetes can be deduced, it must be ascertained that there is a considerable quantity, and not a mere trace, of sugar in the urine; secondly, and especially, that its appearance is not temporary, but persistent; and thirdly, that there is a less or greater increase in the volume of the urine.

A more recondite diagnosis than this, is at present rarely possible: but it is to be hoped, that the time is not very far distant, when we shall be able to indicate the seat of the initial lesion in each case, and to refer it to a cephalic, spinal, sympathetic, hepatic or other category, as the symptoms or previous history may point out.

Prognosis.—The general prognosis is highly unfavourable: the large majority of the cases terminate fatally. A not inconsiderable number, however, recover completely; and many more attain to a state of conditional amelioration—that is, an amelioration which is conditional on the observance of a certain diet and regimen.

The special prognosis depends on a variety of circumstances, of which the following are the more important. The younger the patient, the less hope of ultimate recovery. All the cases under twenty, which I have seen, have eventually succumbed. In persons advanced in years, the appearance and persistence of sugar in the urine is a far less serious affair: it may continue for many years in oscillating quantity with fair preservation of health. It is a curious circumstance that diabetes in corpulent persons is very markedly less formidable than in those of spare habit. Saccharine urine without diuresis is far less serious than when the urine is abundant. *Cæteris paribus*, the longer the disease has existed, the more unfavourable the prognosis; *cæteris paribus*, also, the greater the general severity of the symptoms, the less is the hope of amendment. The ascertained cause of the disease also affects the prognosis. Cases which can be traced to mental anxiety and traumatic lesions appear to be somewhat more hopeful than those for which no tangible cause can be assigned.

The presence of albumen in the urine, of thoracic or intestinal complications, are fatal signs. The existence of permanent amblyopia, or cataract, is a very unfavourable indication. Such cases generally terminate fatally within six or twelve months, and, so far as is now known, always eventually: that is, they are essentially incurable cases, though some of them survive many years.

The results of treatment furnish important data for estimating the gravity of the prognosis. A very favourable circumstance is the disappearance of sugar from the urine when saccharine and starchy matters are withdrawn from the diet. Even great diminution without total disappearance of sugar is a hopeful sign. On the other hand, the persistence of sugar in quantity on a purely animal diet is a sign that the disease is confirmed and far advanced. A moist, perspirable skin, a fair appetite, a stationary condition, are all favourable signs.

It must be remembered, that when, by treatment, the disease has been brought apparently to a stand-still, a diabetic patient still holds his life by a very frail tenure. To use the expression of Dr. Prout, persons with confirmed diabetes, though apparently in fair health, exist as it were on the brink of a precipice. A little undue exposure to wet or cold, an unusual bodily exertion—trifles to the healthy—may excite inflammatory complications which prove rapidly fatal.

TREATMENT.

The seat and nature of the primary lesion is, as we have seen, nearly always concealed; and we know almost nothing of what may be called a radical treatment of diabetes. But the more prominent symptoms—thirst, inordinate appetite, emaciation, and the copious diuresis, are unquestionably dependent, in great part, on the accumulation of sugar in the blood, and the imperious necessity for its removal. A clear indication for treatment, therefore, is to diminish this accumulation. In our ignorance of any direct means of checking the formation of sugar in the body, we resort to the indirect method of withdrawing sugar and amylaceous substances (which are easily converted into sugar in the *primæ viæ*) from the dietary. We endeavour further to combat any existing disorders of the skin, stomach, bowels and other internal organs, and to allay certain troublesome symptoms which arise in the course of the disease. By means of a regulated diet, clothing, and habits of life, invaluable help can be rendered to diabetic patients: sometimes so as to open the way to perfect recovery: often, nay generally, so as to relieve suffering and prolong life.

Diet.—The plan to be pursued is to withdraw as completely as possible, but not too suddenly, all saccharine and amylaceous articles—the chief of which are bread and potatoes—from the diet, and to replace them by appropriate substitutes from the vegetable kingdom, and by animal food.

It is well known that human life can be sustained in perfect vigour on a purely animal diet. The inhabitants of the arctic regions subsist exclusively on the flesh and blubber of seals, fish, and such produce of the chase as the climate affords. The fur-hunters of British America exist for many successive months, leading a life of great muscular activity, on a flesh diet alone. But in our more settled communities the use of bread and potatoes is almost a second nature, and deprivation of them is, to the great majority of individuals, an almost unendurable hardship. To obviate this difficulty several substitutes for bread have been contrived which are of very great value in the management of diabetes.

In the choice of substances from the animal kingdom, the only doubtful or forbidden articles are milk, honey, and liver. Butcher's meat, cheese, butter, fat and oil, poultry, game, eggs, fish, may be used freely in any form. Broths, soups, and jellies (prepared without meal or sugar) are also permissible *ad libitum*. Milk, which contains considerable proportions of a saccharine substance, should as far as possible, be replaced by cream. Milk however is much less deleterious to diabetic patients than might have been supposed. In a girl with confirmed diabetes I made the following trial of the effect of milk. For four weeks she was fed on animal flesh and bran cakes; during the succeeding four weeks three pints of milk daily were added to this diet; and for three weeks subsequently, the milk was withdrawn. The annexed table shows the exact results of the treatment.

	Average daily quantity of urine.	Average quantity of sugar daily excreted.	Increase of weight.
Meat diet, and bran cakes; for four weeks	55 oz.	897 grains.	5 lbs.
Meat diet, bran cakes, and three pints of milk; for four weeks	49 oz.	1260 grains.	5 lbs.
Meat diet, gluten bread, and cabbage; for three weeks	41 oz.	1020 grains.	7 lbs.

The patient continued to gain weight and to improve in her general condition under the use of milk, although the density of the urine and the excretion of sugar somewhat increased. A limited supply of milk may therefore be allowed.

Liver, as found in the butcher's shops, contains a considerable quantity of sugar; it also contains amyloid substance, which is changed into sugar by the saliva, and pancreatic juice. Liver is therefore to be avoided by diabetic patients. The edible mollusks—oysters, cockles, mussels, etc.—are also improper, on account of the large quantity of amyloid substance contained in their enormous livers. For the same reason, the "pudding" of crabs and lobsters is objectionable.

The prohibited articles among vegetables are much more numerous and important, and the substitutes less perfect and more difficult to find.

The oldest substitute for bread is the "bran cake." The husk or bran of wheat consists of lignin and an albuminoid substance, and is quite devoid of starch. When this is washed and ground it may be made up into a rude imitation of bread with butter and eggs, and constitutes a valuable addition to the diet of a diabetic patient.*

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* The best formula for bran cakes is the following, supplied by Dr. Camplin :—

"*Formula for Bran Cakes.*—Take a sufficient quantity (say a quart) of wheat bran, boil it in two successive waters for a quarter of an hour, each time straining it through a sieve, then wash it well with cold water (on the sieve), until the water runs off perfectly clear ; squeeze the bran in a cloth as dry as you can, then spread it thinly on a dish, and place it in a slow oven ; if put in at night let it remain until the morning, when, if perfectly dry and crisp, it will be fit for grinding. The bran thus prepared must be ground in a fine mill and sifted through a wire sieve of such fineness as to require the use of a brush to pass it through ; that which remains in the sieve must be ground again until it becomes quite soft and fine. Take of this bran powder 8 oz. (some patients use 4 oz.), the other ingredients as follows, three new-laid eggs, 1½ oz. (or 2 oz. if desired) of butter, and about half a pint of milk, mix the eggs with a little of the milk, and warm the butter with the other portion ; then stir the whole well together, adding a little nutmeg and ginger, or any other agreeable spice. Bake in small tins (pattipans), which must be well buttered, in a rather quick oven for about half an hour. The cakes, when baked, should be a little thicker than a captain's biscuit ; they may be eaten with meat or cheese for breakfast, dinner, and supper : at tea they require rather a free allowance of butter, or may be eaten with *curd* or any of the soft cheeses.

It is important that the above directions as to *washing* and drying the bran should be exactly followed, in order that it may be freed from starch, and rendered more friable. Mr. White, of Holborn, who made my mill, and was subsequently employed by others, attempted to grind the bran for them, and failed, from not washing and drying the bran, which, in its common state, is soft, and not easily reducible to fine powder. In some seasons of the year, or if the cake has not been well prepared, it changes more rapidly than is convenient. This may be prevented by placing the cake before the fire for five or ten minutes every day."—(Camplin on Diabetes Appendix). These cakes may be had from Blatchley, 362, Oxford Street, London. The mills for grinding the bran are made by Gallop, 119, Cheapside.

Another important substitute is Bouchardat's "gluten bread." This is prepared by washing out the starch from wheaten flour, and working up the remaining gluten into loaves and cakes. This bread is manufactured on a large scale in France, with the aid of powerful machinery for inflating the dough with compressed air, or carbonic acid gas. It forms a light and elegant, and by no means unpalatable bread. Gluten is also ground down into a meal and may be used for thickening broths and making puddings.* These preparations are not quite free from starch; all the samples examined by me showed an intense blue coloration with iodine.

Dr. Pavy has recently introduced rusks and biscuits prepared from the starchless meal of the sweet almond.† These are more expensive than the foregoing: but I have found that patients relished them as a change.

None of these substitutes are as palatable as ordinary bread: but they are of great service; and may be used one after the other, as the patient's inclination may direct. When none of these can be had, or when the patient refuses all three, as is sometimes the case, recourse may be had to "torrefied" bread. Thin slices of ordinary bread are toasted before the fire until they are deeply and thoroughly browned —almost blackened. The starch and gluten are in great part destroyed by the heat, but the hungering diabetic relishes greatly the charred remnants when well-buttered and eaten with other articles.

Rice, tapioca, sago, semola, maccaroni, and vermicelli all contain great abundance of starch, and are therefore inadmissible. Apples, pears, gooseberries, currants, plums, oranges, and all sweet fruits, are likewise pernicious from the quantity of sugar which they contain.

In place of potatoes, turnips, carrots, parsnips, beans, and peas—all of which contain starch or sugar—substitutes may be found in green vegetables—cabbage, endive, spinach, broccoli, Brussels sprouts, lettuce, spring onions, water-cress, mustard and garden cress, and celery.

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* Gluten bread and other gluten preparations made after Bouchardat's formula, are supplied by Van Abbott & Co., Howford Buildings, Fenchurch Street, London. They may also be had of Jewsbury and Brown, Market Street, Manchester.

† Almond rusks and biscuits are supplied by Hill, Bishopsgate Street, London.

There does not seem to be any real advantage in forcibly curtailing, beyond a moderate degree, the fluids taken by diabetic patients. The volume of the urine and the separation of sugar may be temporarily reduced by this means, but the general distress increases, owing to the more intense impregnation of the blood with sugar. Prout recommends that all fluids be given in a tepid state, as they thus allay the craving for liquids more effectually than when taken cold.

In the way of beverages, tea and coffee (without sugar) may be used. Pure glycerine (Price's) may be freely employed as a sweetener instead of sugar. Chocolate made with gluten meal, and soda water, may also be used. The free use of wine and spirits which is especially recommended by Bouchardat as a part of the diabetic regimen, is of more than doubtful propriety. Exact observations do not support Bouchardat's views, which are based on theoretical grounds. Griesinger found that the use of red wine, to the extent of a bottle and a-half or two bottles per day, strengthened with two ounces of rectified spirit, increased considerably both the quantity of urine and the excretion of sugar. In a second observation by the same author, the use of alcoholic drinks caused, in addition to the above effects, a copious diaphoresis of saccharine sweat. The observations of Garrod, Camplin, Rosenstein, Siemssen, and Heller, are also unfavourable to the free use of beer, wine, and spirits. They should therefore be used sparingly. The best are those which are most free from sugar, namely, dry sherry, claret, bitter ale, brandy, and whiskey. Those to be avoided are port, sweet and effervescent wines, sweet ales, porter, rum, and gin.

The use of acid drinks, and especially dilute phosphoric acid, has been highly spoken of in some quarters. Griesinger reports unfavourably of their effects. He pushed dilute phosphoric acid to the extent of an ounce daily. At first, and under the smaller doses, the patient seemed to do very well ; but after ten days, and with the full quantity, the volume of the urine and the proportion of sugar slightly increased, and the general state of the patient grew worse. I have frequently employed bitartrate of potash water for the purpose of allaying thirst, with good effect.

The patient should be clad from head to foot in flannel, in order to encourage the action of the skin, and defend the patient from the chilly sensations so common in this complaint. A warm bath once or twice a week is also very grateful to the patient, and abates the harsh, arid condition of the skin.

The results obtained from the dietetic treatment, differ a good deal according to the intensity of the disease, and the length of time it has existed. The following records illustrate the varying degrees of amendment which may be anticipated in confirmed cases. In the first two cases the patients were permanently cured. The third and fourth cases were inveterate, and, strictly speaking, incurable; in these the quantity of the urine was restored (temporarily at least) almost to its natural limits, and the patients gained flesh and strength in a very remarkable degree: sugar, however, still persisted in the urine, and any deviation from the prescribed regimen was sufficient to reawaken the diabetic symptoms in full intensity. In the fifth case, not much more than a temporary arrest of the downward course was attained, and this was speedily followed by a resumption of the untoward march to a fatal termination.

CASE I.—C. R., æt. 39, of a corpulent habit, came under my care in Oct., 1861. The urine amounted to eight pints a day; specific gravity, 1040; it contained a large quantity of sugar. He had lost much weight, but was still in full flesh. The ordinary symptoms of diabetes were present in moderate intensity. C. R. had been dyspeptic for about fourteen years, though his habits had been, in every respect, temperate. He underwent the operation of lithotomy when a child. For the last two years, he had perceived, that he gradually lost flesh, had an unusual thirst, and frequent desire to pass water. During this period, he had to get up three or four times each night to empty the bladder. Latterly the ankles had begun to swell. Most of the teeth were carious, and the gums loose and spongy. For two months the patient was treated as an out-patient of the Royal Infirmary, and enjoined to avoid saccharine and amylaceous articles of food. It was found that the treatment was carried out very inefficiently; he was therefore admitted as an in-patient on Dec. 4, 1861.

For a week, he was abandoned to the ordinary mixed diet of the hospital. During this week he voided daily on an average 160 ounces of urine: specific gravity, 1035—1040; mean daily excretion of sugar, 5680 grains. He was then put on a diet of animal substances, with cabbage and bran cakes.

In the week succeeding the change of diet, the mean daily discharge of urine fell to 60 ounces: specific gravity, 1022—1026. The sugar fell on the third day to 134 grains, on the fourth to 116 grains, and at the end of the week to 48 grains. In the second week the urine fell to its natural volume and density, and the sugar was reduced to a mere trace. This trace persisted for six weeks, when it suddenly disappeared. The patient gained weight at the rate of three pounds a week. He was then made an out-patient, and directed to continue the restricted diet.

A trace of sugar reappeared and disappeared, from time to time, for several months, but ceased altogether in about eight months. He gradually resumed the moderate use of ordinary bread, and came to show himself at intervals. I saw him last in February, 1865. The urine was found perfectly free from sugar, and the general health and embonpoint were completely restored.

Case II.—T. H., a very stout, florid-complexioned man, 34 years of age, who weighed, when in health, over sixteen stone, came under my care Sep. 19, 1864. He stated that in the previous July, when the weather was very sultry, he perspired very freely, and drank large quantities of cold effervescing beverages.

From this period, a violent thirst and frequent desire to void large quantities of urine, tormented him. He lost weight to the extent of about 40lb.; he was voraciously hungry, and his strength gradually declined.

When first seen by me, the daily discharge of urine amounted to eight pints: specific gravity, 1048; sugar, 7540 grains per day. The general symptoms were mild. The tongue and skin were moist, the teeth sound, the gums only slightly spongy. He complained of incessant thirst, inordinate appetite, pain in the back, and feebleness.

He was put on a restricted diet on Sep. 22, and observed the directions given to him with the most praiseworthy strictness. He was allowed bran cakes, butter, fresh meat, eggs, cabbage, tea and coffee sweetened with glycerine, *ad libitum*. He was cut off from potatoes at once, and after two days, likewise from ordinary bread, and limited entirely to the articles above enumerated. A warm bath was administered every evening, and a pill containing half a grain of opium and one grain of sulphate of iron was given three times a day.

On the third day great improvement had taken place. The urine was reduced to 50 ounces: specific gravity, 1028; sugar, 210 grains.

For two days the patient's condition remained in every respect stationary; but on Sep. 28th he did not feel so well. The urine had fallen to 20 ounces, and the sugar to a very small quantity; the pulse was 98, tongue furred, and a degree of pyrexia prevailed. He sweated profusely after the baths; and some hæmorrhoids to which he was subject, became very painful, the bowels being confined.

This disturbance was attributed partly to the somewhat too sudden revolution in the diet, and partly to the constipating effects of the pills.

On Sep. 29, the pills were withdrawn, the baths were administered every other evening, instead of daily, a little ordinary bread was allowed, a dose of castor oil administered, and the patient directed to keep his bed.

In a few days this disturbance subsided, and the restricted diet was again rigidly enforced. Rapid amendment set in; the urine returned to its natural quantity and density; the sugar gradually diminished, and on Oct. 17, it had entirely disappeared from the urine.

The restricted diet was adhered to for another fortnight, and then a gradual return to the use of ordinary bread was permitted, the urine being carefully examined for sugar from time to time, but none found.

At the beginning of 1865, the bran cakes were discontinued; ordinary bread was allowed freely, and a small portion of potatoes. At the end of January, all restrictions on the diet were withdrawn. The patient had now reached almost his original weight of 16 stone, and felt himself in every respect perfectly well. He was last seen on July 25, 1865. The urine was found perfectly free from sugar.

In the first of these instances, a confirmed but mild case of diabetes, of two years' standing, was perfectly and permanently cured by the dietetic treatment in about eight months. In the second instance, diabetes of three months' standing was completely cured in less than a month. Recoveries so complete as these are, unfortunately, rare. The two following are examples of the *conditional* amelioration, which may be commonly attained, even in severe cases:—

CASE III.—E. H., a well-grown girl of 16, a factory hand, had been diabetic for three years. She was admitted into the Manchester Infirmary, March 26, 1860.

The disease was uncomplicated, and exhibited in great severity the outward signs of diabetes in an advanced stage. There was a harsh, dry skin; a tongue like a piece of broiled ham, and deeply furrowed; abdominal pains, constant drowsiness, consuming thirst, gross appetite, dry scurfy skin, and great emaciation.

For a fortnight after admission, she was put on the common diet of the hospital, which includes a liberal allowance of meat, potatoes, and bread. The state of the urine, during the last six days of this fortnight, was as follows:—Mean daily discharge, 210 ounces; mean daily excretion of sugar, 10,400 grains; average density, 1042. Her weight was 80 lbs.

The diet was then changed. Milk and all vegetable compounds were withdrawn: instead, she was allowed an unlimited supply of eggs, fresh meat, and beef-tea. The patient did not, however, observe my directions strictly, but obtained, and surreptitiously consumed, certain quantities of oranges, sugar, and treacle-toffy. Nevertheless, a remarkable improvement in her condition took place. At the end of eleven days, the mean results since the change of diet were:—Daily discharge of urine, 70 ounces; sugar, 1860 grains; average density, 1034. Weight, 81 lbs. The general health was also much ameliorated; the skin was softer, the tongue less fiery, the thirst and appetite allayed.

On April 21st, bran cakes were added to the animal diet, and greatly relished by the patient. From this date to May 16th—a period of 26 days—no further change was made. The results are shown in the following table. I have divided the period into weeks, for the purpose of displaying the gradual progress :—

	Average daily quantity of urine. ounces.	Range of density.	Sugar each day. grains.	Weight. lbs.
First week	54	1025—1033	1160	81
Second week	67	1021—1031	970	84
Third week	51	1022—1035	870	85
Fourth week	49	1019—1035	690	86
Entire period	55	1019—1035	897	

With this increase of weight, her general condition had improved; the tongue had become pale and moist, but it was still mapped on the surface, and unnaturally smooth.

On the 16th of May, milk was added to the previous diet: the results are given in a preceding page (see page 189). On June 12th, milk was again withdrawn, and gluten bread substituted for bran cakes. Cabbage was also allowed with dinner. The flow of urine on this diet averaged 41 oz., and the sugar 1020 grains per day. The body-weight went on increasing to 98 lbs. Her general condition was now, at the end of eleven weeks of treatment, such, that an unprofessional person would have pronounced her cured. The outward signs of diabetes had disappeared; the skin was restored to its natural softness; the thirst and appetite were no longer inordinate; the flow of urine was reduced within the normal compass. The patient had gained 18 lbs. in weight; she slept soundly, had neither pain nor ache; her strength was so far restored, that she was able actively to assist the nurses in the work of the wards. She came from a distant town, and her history after leaving the infirmary is unknown to me.

Case IV.—W. A., a factory hand, æt. 30, was admitted as an out-patient, October 12, 1859. He presented the usual appearance of diabetes in full career. The disease was uncomplicated, and had existed about a year. The quantity of urine varied from 10 to 15 pints daily, and its density averaged 1044. The patient was directed to observe a restricted diet; and a pill containing a grain of opium, with a quarter of a grain of sulphate of iron, and half a grain of quinine, was ordered three times a day. This treatment was continued—the doses of opium being gradually increased—for seven months. A marked improvement took place; the diabetic symptoms abated considerably; the tongue became moist; the urine fell to five and six pints daily, with a specific gravity of 1040. The sugar averaged 4400 grains. He gained strength and some weight, and was able to resume his occupation for a time. As his condition appeared stationary, he was made an in-patient on May 8th, 1860. On his admission, all medicines were discontinued, and the patient was allowed the mixed diet of the house. The effect of this change was a sudden re-appearance of all the untoward symptoms, with a sense of great debility, and an alarming cough. The condition of the urine was as follows :—Daily discharge of urine, 205 oz. ; sugar, 7400 grains ; average density, 1042. Three days of this freedom from treatment had forced him to keep his bed.

I now gradually withdrew all amylaceous substances, and substituted meat, fish, eggs, and beef-tea. He was also allowed eight ounces of brandy daily. After the change was completed, the diet was absolutely devoid of starch and sugar. Under this diet, the urine altered greatly for the better. During the first week of the restricted diet, the daily discharge of urine was 61 oz. ; daily excretion of sugar, 928 grains ; average density, 1032. The general symptoms also improved, but not in proportion to the amelioration in the condition of the urine.

A second week of the same treatment brought down the urine to—daily discharge, 56 oz. ; daily excretion of sugar, 658 grains ; average density, 1028.

I was now met with the difficulty which so many have encountered in pursuing this treatment ; namely, a total failure of the appetite, and consequent alarming depression of all the vital powers. To obviate these untoward events, the patient was allowed bran bread and the free use of green vegetables—cabbage, lettuce, and water-cresses. A grain of opium was also given three times a day. The diet was therefore still starchless, and almost entirely devoid of sugar. Decided amendment followed this change, and in a few days the returning strength and cheerfulness kept pace with the improved appetite and increasing weight.

During the remainder of his stay in the infirmary, a period of two months, no further change of importance was made in the diet or medicine. The patient's weight on admission was 97 lbs. ; but it rapidly sank in the first few days, and at the end of three weeks it was only 91 lbs. From this time onward, however, the weight began to increase, and it went up gradually to 105 lbs., which point it had reached the week of his discharge.

The state of the urine for the last two months was remarkably constant. The daily discharge varied from 40 to 60 oz. ; the daily excretion of sugar, from 800 to 1000 grains ; the average density, from 1030 to 1033.

The excretion of sugar ruled higher than when the diet was exclusively animal. This I attributed to the improved appetite, which enabled the patient to take more nourishment, rather than to any untoward influence exercised by the green vegetables.

I might greatly multiply examples of this class ; but it will be more useful, to illustrate the less fortunate results for which the practitioner must also be prepared.

Case V.—E. B., a niece of the patient C. R., who made so good a recovery, was admitted into the Royal Infirmary in December, 1862. She had been diabetic for 16 months ; and suffered from excessive thirst, voracious appetite, and great emaciation. The tongue was glazed, skin harsh and dry. There was no complication. The urine amounted to 15 pints a day, and contained over 10,000 grains of sugar, when she lived on a mixed diet.

She remained in hospital two months ; and was gradually limited to a diet of animal flesh, with eggs, cabbage, and bran bread. On this diet she slowly gained three pounds in weight, and improved sensibly in her general health. The urine, however, never fell below five pints; usually it oscillated between seven and eight pints, with a specific gravity ranging from 1030 to 1040, and a daily excretion of sugar of 4450 to 7420 grains.

After leaving the Infirmary, she speedily relapsed, gradually grew worse, and died in March, 1863, in the Withington Workhouse.

Much discredit has been thrown on the dietetic treatment, by a slovenly and incomplete manner of carrying it out. It requires most vigilant watching to keep guard against the admission of forbidden articles. The patient's own craving for them is often too much for his resolution, and most artful deceits are practised on the medical attendant. This is especially the case at the beginning of the treatment. After awhile, the patient perceives from his own experience, the importance of abstaining, and the desire for the forbidden articles diminishes very notably after the lapse of some weeks. Amylaceous compounds, too, are often unwittingly administered by the attendants. Starchy matter is never absent from the cook's hand; it enters, in one guise or other, into almost every dish.

Then there arises the other difficulty—the rejection by the stomach of the restricted diet. This difficulty is perhaps made too much of. A skilful selection and frequent change of articles of diet, usually suffices to reconcile the digestive organs. The field of selection among admissible articles is so wide that, in private practice, the practitioner's resources are inexhaustible. Among hospital patients, however, the embarrassments on this score are very serious.

There are cases of such severity, that not even a temporary amendment can be obtained by the dietetic treatment. I have known more than one such instance in children under ten years of age, in whom the disease ran a rapid course, and proved fatal in a few months. There are also a certain number of chronic cases in which the dietetic treatment proves unsuitable, and hastens rather than retards the final catastrophe. These are for the most part long standing cases—cases, perhaps, which have been benefited at a previous epoch by that treatment. In two of my infirmary patients, who were re-admitted to the benefits of the charity after an interval of several months, a much more decided amelioration followed the dietetic treatment, during the first period of their stay, than during the second.

The sugar-forming vice of the diabetic system appears at first (and throughout in the milder cases), confined to saccharine and amylaceous articles of food; but as the disease becomes inveterate, the assimilation of the albuminous principles is more and more affected, until at length, these yield sugar almost as readily as the former. Griesinger found in a case of this kind, on strict flesh diet, that three-fifths of the albuminous materials re-appeared in the urine as sugar. When matters have come to this pass, it is not to be wondered at, that the patient no longer derives benefit from a restricted diet, which he can only use sparingly, and almost with disgust, and that he should, on the whole, find himself in a better position when abandoned to ordinary mixed fare, which he can consume in abundance, and with relish. Experience is imperative on this point. When a flesh diet, judiciously eked out by appropriate substitutes for bread and potatoes, fails to ameliorate the general condition, it should not be too obstinately persisted in after a fair trial. The practitioner should give way first with regard to bread, and hold out longest against potatoes. No inflexible and universal rule can be laid down respecting the diet of diabetic individuals, under all circumstances and in all stages of the complaint. Cases will occur, in which the power to take a plentiful supply of a mixed diet, more than compensates for the increasing thirst and freer discharge of urine and sugar. I have also noted that some of the milder types of this disease, in which saccharine urine is unaccompanied with diuresis, are made worse by a too restricted diet (see appendix to this chapter).

Medicinal Substances in Diabetes: Supplementary Means.—Some of these are employed under the impression that they possess a really curative power in this disease: others are resorted to, simply as adjuvants, to combat some particular symptom.

The inquiries hitherto made on the supplementary means—medicinal and other—employed in the treatment of diabetes, are mostly vitiated, by an insufficient separation of their effects, from those of the restricted diet, which is usually conjoined therewith, and a want of attention to the clinical grouping of the cases. A number of remedies have been extravagantly lauded on diverse hands, and have in this way attained an ephemeral reputation; but when tried by accurate observers, they have proved to be absolutely inert. Unless the points just indicated are kept in view, only misleading conclusions can be drawn from any inquiries on this subject. It is quite possible, that remedies which have proved powerless in inveterate cases, may be of real service in milder examples of a different type, or in the earlier stages of the disease. A complete revision of the supplementary means of treating diabetes is loudly called for. It may be taken for granted, that the general basis of all treatment of diabetes, must be the dietetic restrictions already described. Other means must be studied with a clear understanding of their supplementary and subordinate place.

Opium.—This narcotic is unquestionably of great use in the treatment of diabetes—not from its direct influence on the course of the disease, but from its anodyne properties. If no restriction be placed on the diet, opium in doses of from 6 to 20 grains a day, has always, in my experience, had the power of reducing the flow of urine by about one half; that is to say, of bringing it down to five or eight pints, and this without increasing its density. But notwithstanding this amelioration in the state of the urine, the downward progress of the disease is not arrested; and the effect of the drug seems attributable to its deadening influence on the appetite, rather than to a specific power of checking the formation of sugar. When opium is given to patients under a restricted diet, it does not exhibit the least power of lessening the flow of urine or the excretion of sugar. Its value depends on its power of inducing sleep, and of allaying the dolorous sensations, and irritability, which constantly torment diabetic patients.

There is great tolerance of opium in confirmed diabetes. Doses of 2, 3, and 5 grains, three times a day, are generally borne without the production of any appreciable narcotism.

Alkalies.—Alkaline substances have been especially recommended by Miahle, on account of their supposed power of favouring the oxidation and destruction of sugar in the blood.

These theoretical views are now overthrown. In two of my patients, I made a trial of full doses of the bicarbonate of potash. One of them was on a mixed ordinary diet, and the disorder was far advanced. The urine was rendered alkaline for ten days without in any way altering the excretion of sugar, or the general condition. In the second case, the patient was on a restricted diet. She took for a fortnight 320 grains of the bicarbonate daily, in divided doses ; the urine was thereby rendered freely alkaline. During the week preceding the alkaline treatment, 1160 grains of sugar were excreted daily. In the first week of the alkaline treatment 970 grains a-day were separated, and in the second week 870 grains. In the week following the withdrawal of the alkali, the sugar amounted to 690. This observation tends to show that the alkali had no appreciable influence on the excretion of sugar. I have not encountered any difficulty in rendering the urine alkaline in diabetes, as Dr. Pavy seems to have done.

Rennet and *Pepsine* have, of late, been vaunted in such terms of confidence, as to raise hopes which are not destined to be realised. The most remarkable results, obtained from rennet, are those published by Dr. James Gray. He states that of twenty-seven persons treated, seven recovered. This is an example of most rare success, and it is to be regretted that the cases are not reported with that exactitude and detail, which are desirable on such debateable ground. In all of them a rigid adherence to animal diet and bran bread was insisted on ; and it seems more than probable that the amendment in each case was due to the restricted diet rather than to the rennet. Dr. Nelson, of Birmingham, extols the same remedy. His cases do not seem to have been severe ones ; and the diet was regulated in at least some of them. The reports are much more imperfect than those of Dr. Gray.

I gave rennet a resolute trial in one confirmed case. It was prepared in the manner recommended by Dr. Gray, and given in doses of two table-spoonfuls three times a-day. The patient took it for more than two months, conjoined with a rigidly restricted diet. During this period he improved, and gained 5 lbs. in weight. But he was improving just as rapidly before he began the rennet, and the daily excretion of sugar had not in the least diminished during its use. Griesinger, in two cases, accurately observed, found even a slight increase of sugar during the use of rennet. Other trustworthy reports are equally unfavourable.

Parkes* and Leubuscher† found pepsine useless.

I conceived that it was worth a trial, whether some of the substances which act powerfully on the nervous system, might not exercise a beneficial effect in diabetes. With this view, I exhibited strychnia and belladonna, in gradually increasing doses, until their physiological effects began to be perceived. But not the slightest influence on the excretion of sugar could be discovered during their use.

In the present state of knowledge, we are forced to the conclusion, that no known medicament possesses the least power of directly checking the sugar-forming vice in diabetes. There is, however, a large field for the operation of adjuvant remedies, employed simply for their ordinary therapeutical effects.

The obstinate constipation, which commonly prevails in diabetes, must be corrected by a regulated use of castor oil, seidlitz powders, or the ordinary rhubarb and magnesia mixture. Anodynes are called for to subdue pain, nervous exhaustion, restlessness, and insomnia. Dyspeptic symptoms are to be combated by alkaline tonics: and for this purpose I know of no better combination than the bicarbonate of potash in infusion of calumba, with hydrocyanic acid. The poverty of the blood and the progressive emaciation, are best combated by long courses of iron and cod-liver oil. I have already spoken of a solution of bitartrate of potash, as the best means of directly allaying the thirst. When the craving for food, and sense of sinking at the epigastrium are troublesome, a pill containing two or three grains of assafœtida, administered twice or thrice a-day, gives most striking relief.

Diabetic patients often reap considerable benefit from change of air, and a sojourn at watering-places. The Bristol Hotwells, Vichy, and Carlsbad waters have obtained some celebrity for their utility in diabetes. In milder cases, sea-bathing may be recommended in moderation in the hot season of the year.*

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* On the Composition of the Urine, p. 356, note.

† Arch. f. Path. Anat. B. xviii., 119.

Saccharine treatment of diabetes.—Piorry conceived the odd idea, that the main evils of diabetes depended on the loss of sugar through the kidneys, and that, by compensating this loss by administering sugar internally, these evils could be overcome. Dr. W. Budd, of Bristol, followed up Piorry's lead, and administered from 5 to 8 ounces of sugar and 4 ounces of honey to two diabetic patients, with great benefit. Ordinary mixed diet (excluding potatoes) was conjoined. These results provoked new trials of this treatment by Dr. Burd, of Shrewsbury, Dr. Sloane, of Leicester, Dr. Bence Jones, and Griesinger, but with results so decidedly unfavourable as to leave no doubt of the inutility of the practice. A full *resumé* of the results of the saccharine treatment of diabetes may be found in a paper by the author in the *Brit. Med. Journ.* for November, 1860.

APPENDIX.

Milder Types of Diabetes.

The cases brought together under this heading are somewhat miscellaneous; and they do not present those marks of uniformity, which are required to constitute a homogeneous pathological group. They are separated from classical diabetes by certain broad distinctions, of clinical importance; but they exhibit among themselves, certain disagreements which make it evident that they represent more than one type of disease.

From ordinary, or classical diabetes, these milder types are distinguished, by all or most of the following signs:—absence of a fixed tendency to a fatal termination; absence, or only moderate degree, of thirst, voracity, and emaciation; slight or temporary increase in the quantity of urine; transitory duration ; amenability to treatment ; slight, moderate, or intermittent glycosuria.

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* Bouchardat, in a recent memoir, speaks in the highest terms of enforced exercise and gymnastics for diabetic patients. See Annuaire de Therap., 1865, p. 291.

The greater number of these cases fall within one or other of the three following groups, to each of which illustrative examples are appended :—

GROUP I.—Urine persistently saccharine ; density 1030 to 1043 ; diuresis absent, or very moderate ; no excessive thirst or appetite ; moderate conservation of strength and flesh ; stationary condition.

CASE I.—Mr. B., a manufacturer, æt. 45, thin, but not markedly emaciated, able to attend to his business, consulted me May 14, 1861. His health had been feeble for six months. He complained of weakness, loss of appetite, and restlessness. The urine had never exceeded four pints, and usually did not exceed three pints, in the twenty-four hours. The specimen sent to me for analysis had a density of 1042, and contained 7·2 per cent. of sugar. There was no inordinate appetite or thirst ; the skin was moist. The patient had tried a diet composed of animal flesh and green vegetables, but had been unable to adhere thereto on account of the total failure of the appetite.

During the last four years I have seen this patient several times. His condition continues unchanged, both as to the general health and the state of the urine. He is still a valetudinarian, but goes about his business, and observes a diet, only restricted with respect to the use of potatoes.

CASE II.—Mr. F., æt. 50, formerly engaged in business. He consulted me in Nov., 1862, and stated that he had been ailing about three years, suffering from indigestion, lowness of spirits, and loss of strength. A twelvemonth before, sugar had been detected in the urine. The urine had not at any time exceeded three pints in the twenty-four hours. He has never been troubled with thirst ; the skin is usually moist ; there has been slight emaciation. He has tried a restricted diet without any benefit.

Two specimens of urine were handed to me for analysis : one on Nov. 20, 1862, and the other on April 22, 1863. The former contained 7·7 per cent. of sugar, and the latter (which had a specific gravity of 1039) 6·3 per cent. The daily quantity at both dates was three pints. The disorder in this instance appears to have arisen from worry and anxiety connected with business ; but for a period of two years after giving up business he remained in *statu quo*, no treatment appearing to have any beneficial result. Recently he has been in much better health, has recovered his weight, strength, and cheerfulness, and believes himself thoroughly rid of his complaint ; and yet, the urine has now (Feb. 23, 1865) a specific gravity of 1035, and contains 5·7 per cent. of sugar.

CASE III.—Dr. Latham[*] relates a case resembling these in most respects, but differing in its fatal termination. The patient was a gentleman, æt.

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[*] Latham on Diabetes, p. 147.

40, well known in the profession of the law. The urine at no time exceeded a quart, but it was so sweet "that it might easily have been mistaken for syrup." The dietetic treatment was resolutely tried without any good effect: he died with cough, colliquative sweats, and other signs of phthisis.

GROUP II.—Glycosuria, temporary or intermittent: thirst and diuresis moderate, or none; little emaciation and loss of strength; the complaint depending on mental anxiety, blows on the head, or concussion of the spine, and terminating in complete recovery.

CASE IV.—A gentleman, æt. 46, engaged in business, consulted me on March 23, 1862. He had suffered from slight, recurrent, dyspeptic symptoms for more than a year, together with numerous nervous phenomena and loss of rest. During this period, he had undergone great mental stress in connection with the responsibilities of a large manufacturing concern. On two occasions he had been seized with some kind of fit, which, from the description given, appeared to be a bastard epilepsy. In one of these, he had fallen from his horse; but there was no direct injury to the head. At my request a specimen of urine was sent for examination. Its specific gravity was 1035, and it contained 5·2 per cent. of sugar; no albumen or other abnormal ingredient was present. The daily quantity did not exceed three pints. He was put on a moderately restricted diet, and recommended to make arrangements which would relieve him of a large portion of his responsibilities. He continued under my observation for six months. The sugar disappeared in about six weeks, except a trace, which also vanished at the end of four months. His health is now (July, 1865) perfectly restored.

One of the most singular instances of glycosuria, persisting for several months, unaccompanied with any of the symptoms of true diabetes, is related by Griesinger (loc. cit. p. 51).

CASE V.—A medical student had, during a course of chemical instruction, in the year 18—, often examined his urine, and found it in every respect normal. He spent the summer of the succeeding year in Switzerland, and underwent a number of wettings on botanical excursions. Some months later, while in perfect health, the appearance of the urine attracted his attention. On examination it gave an abundant sugar reaction with Trommer's test. He now examined the urine daily, and found the density to vary between 1022 and 1027. The glycosuria persisted throughout the following winter, during which he continued to reside in the same moist and foggy locality. In the succeeding spring, Herr —— returned from Switzerland, and, being much occupied, had no longer any time to bestow on his diabetes; and when, in the course of the ensuing summer, he examined the urine again, he found it totally free from sugar, nor has a trace been found in it since. During the entire period that the urine contained sugar he did not experience a single one of the known symptoms of diabetes.

Group III.—Glycosuria in persons advanced in years; of full habit; moderate conservation of flesh and strength; moderate diuresis; moderate amount of sugar; abundance of uric acid deposits; often gout; sugar sometimes present for years, varying greatly in quantity, sometimes intermitting—termination variable.

Dr. Bence Jones has published an account of a number of cases of this class.[*] Of twenty-nine cases of glycosuria, observed by him in the preceding three years, eleven were above sixty years of age, and six of these were above seventy. He supplies the following analysis of these eleven cases:—

In 2, The disease was intermitting.
„ 6, The quantity of urine was scarcely, if at all, increased.
„ 1, The quantity was increased, but the disease had probably existed for sixteen years.
„ 1, The urine was albuminous, and the diabetic symptoms were very slight.
„ 1 (Above seventy-four years of age), the disease existed in its intensity.

In all the cases save one, the disease was of exceedingly mild character.

Several cases of this kind have come under my notice, of which the two following examples may serve for illustrations.

Case VI.—Mr. A., a surgeon, æt. 60, a tall, stout man, of powerful frame, consulted me June 11, 1863. He had noticed for the last four months an undue frequency of micturition, with a certain languor unusual to him, of which, however, he thought little, until the copiousness of the urine excited his suspicions, and induced him to test it for sugar. This led to the detection of his complaint. He had lost some flesh.

When I examined him, he had a ruddy complexion and an appearance of health; the appetite was moderate; thirst somewhat troublesome; skin moist; he went about his usual business—being in extensive practice in a rural district—with scarcely more fatigue than ordinary. The teeth were extensively decayed. The urine amounted to five and six pints daily. A specimen carefully collected for twelve hours was sent to me for examination. It amounted to 68oz.; specific gravity, 1034; it deposited uric acid copiously, and contained 6 per cent. of sugar, which indicated a total of 1800 grains in half a day.

Mr. A. was gradually put on a restricted diet, with gluten bread. In a week, the urine of twelve hours had come down to 45 ounces: specific gravity, 1035; per-centage of sugar, 6·1; sugar voided in twelve hours, 1190 grains.

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[*] Med. Chir. Trans., vol. xxxvi.

Four weeks later, the urine of twelve hours had diminished to 37 ounces: specific gravity, 1028; sugar 4 per cent; quantity voided in twelve hours, 673 grains. The general condition had also greatly improved; he still adhered to the restricted diet.

I have seen Mr. A. from time to time up to the present date (Feb. 1865). He is now perfectly restored to his original health and embonpoint. The restrictions on his diet have long since been relaxed. He derived considerable advantage from the use of almond rusks and cakes, and from change of air and scene, in the highlands of Scotland.

CASE VII.—Mr. M., a retired solicitor, æt. 72, consulted me Oct. 17, 1863. He was a florid-complexioned, stout, healthy, and vigorous looking man for his age. Until sixteen months ago he had always enjoyed excellent health.

Sixteen months ago he was seized with a low febrile complaint of undetermined character. He kept his bed for two months, and was greatly reduced by it; but he gradually recovered, and went to Buxton to complete his convalescence. Before going to Buxton, he had noticed a sweet taste in his mouth and a certain sweetness of the skin of his hands; and when there, he noticed a great thirst and frequent calls to void urine. With the continuance of these symptoms he became rapidly thinner, and sent for his son-in-law, Dr. H., who examined the urine, and discovered sugar. Dr. H. found the symptoms of diabetes present in moderate intensity; gums spongy; emaciation very considerable; all his embonpoint gone; he was "reduced to a little old man." The urine amounted to six and ten pints a day; and his thirst was so tormenting, that he used to prepare for himself a large jug-full of oatmeal-water and milk, to drink at night.

At this period, he was put upon a strict flesh diet, with green vegetables. Great benefit followed this treatment; and in about two months from the first onset of the diabetic symptoms, he had recovered from the attack, and begun to recover flesh and strength. It was not ascertained, whether the sugar disappeared from the urine, when the other symptoms subsided.

He continued in improved health for five or six months, and regained much of his previous vigour. He then began to suffer from severe lancinating pains about the base of the chest. On account of these he sought my aid.

He complained of intense pain, of neuralgic character, along the course of the lower intercostal nerves. Up to the day before his visit to me, the pain had been limited to the left side, but it had now invaded the right side; and a painful circle embraced him, in a line corresponding to the attachments of the diaphragm. The pain was darting, burning, as if a red-hot iron were drawn round him; it prevailed in paroxysms; but lately the remissions had never been complete; and the pain came forward to the mesial line, and descended into the testicles and penis. Nightly opiates were required to induce sleep. He was very nervous and agitated, especially during the paroxysms, but there was no fever. Tongue clean, pulse quiet, ranging from 65 to 80 (he often counted it himself); heart's sounds were healthy, and there was no hypertrophy. The pain was much increased by motion of the body, as in walking. There was no thirst; the quantity of urine was not increased. Micturition frequent at night; appetite pretty fair.

At my request, he brought me the urine made after dinner on October 18th; its specific gravity was 1030, clear, amber-coloured; it contained no trace of albumen, but as much as 5·1 per cent. of sugar. He was ordered 5 grains of quinine, with some carbonate of iron, and a few drops of laudanum, at night.

Oct. 19.—He brought me the urine made before breakfast; its specific gravity was 1019, and it contained only a trace of sugar. He had passed a much better night than usual.

Oct. 20.—Urine before breakfast contained a trace of sugar: that voided after dinner contained a good deal more.

Oct. 21.—Urine before breakfast was quite free from sugar; that after dinner contained 4 per cent. He still complained of the pain round the chest, but in much diminished degree.

Oct. 25.—Urine before breakfast free from sugar; that passed after dinner contained only 0·8 per cent.

He was put for a while on a partially restricted diet. The urine continued for some days to show traces of sugar after dinner. After this he left town and went to the country, continuing to improve. This gentleman is now (February, 1865) in very fair health for his age; but I cannot state, whether or not the urine contains sugar.

CHAPTER III.

GRAVEL AND CALCULUS.

(Urolithiasis.)

Marcet—On Calculous Disorders. Lond. 1819.
Prout—Nature and Treatment of Gravel and Calculus. Lond. 1821.
Magendie—De la Gravelle. Paris.
Civiale—Traité de l'Affection Calculeuse. Paris, 1838.
Crosse—A Treatise on Urinary Calculus. Lond. 1841.
Catalogue of Calculi in the Museum of the College of Surgeons. Lond. 1842.
Rees—On Calculous Disease. Lond. 1856.
Heller—Die Harnconcretionen. Vienna, 1860.
Leroy d'Etiolles (Fils)—Traité pratique de la Gravelle. Paris, 1863-4.
Beale—Urine, Urinary Deposits, and Calculi. 2nd ed. Lond. 1864.

GENERAL ETIOLOGY.

The deaths from stone, in England and Wales, in the five years ending 1861, amounted to an annual average of 184. It is satisfactory to note that the mortality from this cause exhibits a progressive diminution in the last five-and-twenty years, as may be seen from the following table constructed from the Registrar-General's Reports :—

Mortality from stone, in England and Wales, in four successive quinquennial periods.[*]

In the 5 years	1838—42	the yearly average of deaths from stone, was	297
,,	1847—51	. . . .	232
,,	1852—56	. . . .	216
,,	1857—61	. . . .	184

The cause of this diminution is to be chiefly sought for, in the earlier detection of the stone, and earlier resort to operation, in recent times: perhaps also in the improved diet and water supply of the population.*

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[*] The returns for the years 1843—46, are tabulated differently from the remainder, and cannot, therefore, be included in this table.

Calculous disease is much more fatal (as might have been expected) in the male, than in the female, sex. For every female that died, in England and Wales, in the five years, 1857-61, from the consequences of stone, nearly ten males perished.

More deaths from stone occur at an early *age*, and in the waning years of life, than in the intermediate periods, as is shown by the following table :—

TABLE *showing the number of deaths from stone, at different ages, in the quinquennial period* 1857—61 *in England and Wales—Males only included.*

Under 5 years	55 deaths.
Between 5 and 15 years	61 ,,
,, 15 ,, 25 ,,	32 ,,
,, 25 ,, 35 ,,	24 ,,
,, 35 ,, 45 ,,	40 ,,
,, 45 ,, 55 ,,	59 ,,
,, 55 ,, 65 ,,	130 ,,
,, 65 ,, 75 ,,	262 ,,
75 and upwards	160 ,,

The great fatality of stone above the age of fifty-five is due, not so much to the greater frequency of stone at that epoch, as to its more severe effects on the constitution, and the less favourable results of operation in advanced life. The *frequency* of stone is far the greatest under five years of age; and next between ten and fifteen years. It then diminishes rapidly until the thirty-fifth year. Above this age cases of stone become, again, more and more frequent, until the age of sixty-five. The following table indicates, very exactly, the prevalence of stone at different periods of life. It embraces all the persons who underwent the operation of lithotomy, during given periods of time, at the following hospitals:—Guy's, St. Thomas's, University College, Norwich, Cambridge, Oxford, Birmingham, Leicester, and Leeds.

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* The suburban district of Hulme supplies considerably fewer cases of stone to the Manchester Infirmary, since the pipe-water has replaced the old pump-water supply.

Table *showing the ages of* 1827 *persons who underwent lithotomy at the above hospitals—constructed from statistics collected in Mr. H. Thompson's work on Practical Lithotomy and Lithotrity.*

Under 5 years	473
Between 5 and 15 years	528
,, 15 ,, 25 ,,	157
,, 25 ,, 35 ,,	85
,, 35 ,, 45 ,,	90
,, 45 ,, 55 ,,	156
,, 55 ,, 65 ,,	225
,, 65 ,, 75 ,,	103
,, 75 ,, 81 ,,	10

No countries or climates are altogether free from calculous disorders; but some localities are considerably more afflicted by them than others. Stone and gravel are common in England, France, Teneriffe, Iceland and Egypt.* They are, on the contrary, rare in Sweden and Norway, Styria, and some other parts of the Austrian dominions. In Christiana, 3211 patients were treated in the general hospital during a period of four years, and among them there was only one stone patient. In the hospital of Gothenburg, in Sweden, which contains sixty beds, not a single case of stone was received in fifteen years.†

The climatic conditions favourable to the prevalence of stone appear to vary within narrow topographical limits. Of the eleven registration districts into which England and Wales are divided, the eastern counties of Norfolk and Suffolk furnish the largest proportion of deaths from stone. Next to these come the North Midland Counties. The fewest deaths from stone (as compared to the total mortality) are furnished by Lancashire and Cheshire, and by the South Western Counties.‡

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* The frequency of stone in Egypt is due to the ravages of the Bilharzia hæmatobia, a minute parasite which infests the urinary organs in hot countries—(see Bilharzia).

† Civiale. Traité de l'Affection Calculeuse, p. 580.

‡ The following table shows the proportion of deaths from stone in each of the eleven registration districts of England and Wales, for every 100,000 deaths from all causes, in the five years 1857—61. Males only are included. (Constructed from the Registrar-General's Reports.)

North-Western	45
South-Western	50

CLASSIFICATION OF URINARY CALCULI, THEIR CHEMICAL CHARACTERS, ORIGIN, GROWTH, AND GENERAL CLINICAL HISTORY.

Urinary calculi may be classified, according to their chemical composition, into eight *primary* and one *secondary* species. The primary species are:—1. Uric acid. 2. Urates. 3. Oxalate of lime. 4. Cystine. 5. Xanthine. 6. Urostealith. 7. Bone earth (basic phosphate of lime). 8. Carbonate of lime. The secondary concretion is composed of a mixture of the phosphate of lime and the ammoniaco-magnesian phosphate.

In addition to these, which are composed of normal or abnormal, but strictly urinary, ingredients, two other species are occasionally found in the urinary passages which have an origin extraneous to the urine. These are *fibrine or blood concretions* and *prostatic calculi.*

Urinary concretions always contain, in addition to their proper components, slight admixtures of animal matters, viz., mucus, epithelium, pigment, and, generally also, more or less desiccated blood and pus.

The term "gravel" is given to concretions of small dimensions, which are not too large to be spontaneously voided by the urethra; the larger masses are called "stones," or "calculi."

Calculous formations are said to be *primary*, when they are deposited from the unchanged urine, owing to some inherent vice in its composition; and *secondary*, when the deposit is due to ammoniacal decomposition of the urine in the lower urinary passages.

It is essential to recognise this difference in order to understand the mode of growth of urinary calculi, and the principles which should guide their medical treatment.

South Midland	62
West Midland	63
Northern	67
Yorkshire	75
London	89
Monmouthshire and Wales	91
South-Eastern	99
North Midland	101
Eastern	126

It has been already explained that whenever the urine becomes decomposed and ammoniacal, its earthy constituents are precipitated as a sediment, composed of phosphate of lime and the ammoniaco-magnesian phosphate, often mixed with small quantities of urate of ammonia and carbonate of lime. This is identical with the *secondary* phosphatic deposit on urinary calculi.* Its production is due to the transformation of urea into carbonate of ammonia. Any obstacle which delays the urine in its channels, and prevents its speedy and complete evacuation, tends to bring about this change. The presence of a calculus in the bladder presents a condition highly favourable to the production of ammoniacal urine, and to the precipitation of the secondary phosphatic deposit. Accordingly it is found, that calculi which have been long detained in the bladder, are frequently covered over with a phosphatic incrustation. Indeed, it may be said that this is the proper ultimate stage and last chapter in the history of every urinary concretion, unless its career be cut short by spontaneous expulsion or removal by surgical operation.

The epoch at which the secondary deposit begins to form is quite uncertain, and depends on the concurrence of cystitis. Sometimes small calculi, weighing only a few drachms, are found covered with a thick investment of phosphates; in other instances large calculi, weighing many ounces, are found without any traces of phosphatic incrustation. So long as the urine remains acid the surface of the stone remains free from phosphates, but as soon as the urine becomes freely ammoniacal, the secondary deposit begins to accumulate.

It follows from these facts, that a solvent treatment which may have been applicable in the early existence of a stone, ceases to be so when the urine becomes ammoniacal and a secondary deposit has taken place.

The principal points relating generally to the structure and growth of urinary calculi are embraced in the following propositions:—

1. Calculi may consist entirely of one ingredient, as uric

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* The fetid incrustation which covers public urinals is likewise of similar nature.

acid, oxalate of lime, cystine, &c.; or two or more primary deposits may alternate with each other in the form of layers, so as to constitute an *alternating* calculus.

2. The most common alternations are uric acid and oxalate of lime; but any primary deposit may alternate with any other primary deposit: as cystine with uric acid; uric acid with bone earth; or oxalate of lime and bone earth. The two last cases are however excessively rare. The number of layers composing an alternating calculus is quite uncertain: there may be only three or four, or twenty or thirty. The thickness of the layers varies conversely with their number.

3. A calculus consisting of only one substance has usually a stratified arrangement, and exhibits an indefinite number of concentric layers. Such is usually the structure of uric acid, oxalate of lime, and phosphatic calculi. But sometimes the calculous matter is deposited in vertical lines radiating from the centre. This is the usual structure of cystine calculi. Sometimes one portion of a stone has a radiated, and another portion a stratified formation.

4. Most urinary calculi are divisible into a central portion or *nucleus*, and an outer portion or *body*. There is also not unfrequently an outer investment, or *crust*, of phosphatic deposit.

5. The nucleus may be of the same nature as the body, or differ from it. The nucleus may consist of uric acid, oxalate of lime, or any other primary formation, or it may be a clot of blood, or a mass of mucus; or, lastly, it may consist of some foreign body introduced from without.

6. The determining *causes* of the formation of urinary calculi are still but imperfectly known. The more usual are the following:—(*a*) An excessive proportion of the precipitated ingredient in the urine; (*b*) A too acid state of the urine, which diminishes its solvent power over uric acid and the urates; (*c*) An alkaline state of the urine. If the alkalescence be due to fixed alkali, the bone-earth phosphate and carbonate of lime are liable to precipitation; this is however a very rare contingency in the human subject, though common in the herbivora. If the alkalescence be due to carbonate of ammonia, the secondary phosphates are precipitated; (*d*)

Deficiency of chloride of sodium and the alkaline phosphates in the urine, reduces its solvent power on uric acid (Heller); (*e*) The presence in the urine of an abnormal constituent of sparing solubility, such as cystine or xanthine; (*f*) The accidental presence of a body suitable to form a nucleus, such as a small mass of concrete blood, mucus, epithelium, or an extraneous body, such as a bit of bougie, a piece of bone, a wire or needle, a bit of sealing-wax, and so forth.

OF THE PARTICULAR VARIETIES OF URINARY GRAVEL AND CALCULI.

1. *Uric acid.*—This is by far the most frequent species of urinary concretion. It constitutes probably five-sixths of all renal concretions, and of vesical calculi which have only recently descended from the kidney. As *gravel*, uric acid may be passed in the form of small distorted crystalline agglomerations, or as little spherical bodies, ranging from the size of a poppy seed to that of a mustard seed, or in flattened warty concretions as large as split peas. All these have a yellowish, brownish, or reddish colour. They are derived from the kidney, and may be discharged singly or in numbers at irregular intervals.

When retained in the bladder, they grow into flattened oval *calculi*, sometimes roundish, sometimes elongated like an almond. They vary in colour from a light fawn to a deep brick-red, according to the quantity and nature of the urinary pigment which they contain. Their surface is usually studded with minute tubercles, or mamillations, which are worn into smooth facets if more than one stone co-exist in the bladder. Their weight varies from a drachm to an ounce, but sometimes reaches four or five ounces.

Uric acid calculi possess considerable hardness; their spec. grav. is about 1·5. Uric acid is best recognised by the murexid test, described at p. 36. Its most important properties, from a therapeutical point of view, are its solubility in very weak solutions of the carbonates of potash and soda, and its insolubility in strong solutions of the same, as well as in water and dilute acids.

Pathologically, uric acid is closely related to gout. Hence the frequency of uric acid gravel and stone in the wealthier classes in the middle and later periods of life.

The urine, in the subjects of uric acid calculi, is acid, and often high-coloured, prone to deposits of uric acid crystals and amorphous urates.

The medical treatment of this class of calculi will be described at length in a separate section (see SOLVENT TREATMENT).

2. *Urate concretions.*—The same confusion has existed respecting the composition of these concretions, as respecting that of the amorphous urate deposit. They are usually designated urate of ammonia, but their chemical nature requires re-examination.

They constitute small, soft, agglomerations in the kidneys —rarely in the bladder; and are almost confined to young children. Heller* states that he has found them several times in the kidneys and ureters of sucking infants in the Vienna Foundling Hospital. They formed small irregular clumps, sometimes heaped together into a mass as large as a kidney bean. Heller encountered similar calculi on two occasions in adults. Urate calculi are very rare, and never reach a large size. It may be doubted whether the calculi described by Prout as urate of ammonia were really anything more than fawn-coloured uric acid.

The deposition of spiny clumps of urate of soda in the urinary passages, is not uncommon in the febrile complaints of infants and young children, especially when there is temporary retention of urine. It seems not unlikely that some of these clumps may be retained in the pelvis of the kidney or in the bladder, and become the nuclei of future calculi; and that the excessive frequency of calculi in children is due to this cause (see p. 45).

The urine from which this variety of concretion is deposited, has an acid re-action, and the medical treatment is identical with that of uric acid calculi. The circumstances under which this concretion is deposited, must be carefully distinguished from those, in which urate of ammonia (of undoubted composition) is deposited in an ammoniacal urine mixed with secondary phosphates.

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* Harnconcretionen, p. 134.

Urate concretions are distinguished chemically by their solubility in hot water.

3. *Oxalate of lime or mulberry calculus.*—Oxalate of lime may be discharged as minute concretions, or gravel, from the kidney, or grow to be a stone in the bladder. In the former case the concretions are usually smooth, rounded, greyish dark bodies, resembling hemp-seed.

Vesical calculi of this class are exceedingly hard, and break into sharp angular fragments when crushed by the lithotrite. They are usually of a spherical shape; their surface is tuberculated like a mulberry (Fig. 33), and is usually of a blackish-brown colour. Sometimes however they are oval and smooth, and of a blueish-gray colour.

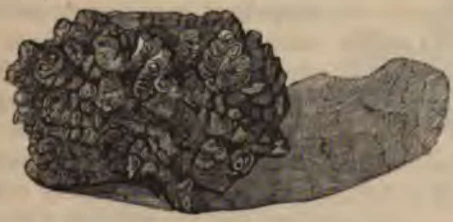

FIG. 33. Oxalate of lime or mulberry calculus.

The nucleus of a mulberry calculus is frequently composed of uric acid; and, conversely (though much more rarely), a uric acid stone may have a nucleus of oxalate of lime. Dr. Beale has further shown, that in the centre of a uric acid nucleus, there is often a microscopic clump of dumb-bells of oxalate of lime.

Calculi composed of alternate layers of oxalate of lime and uric acid, are more common than those composed of oxalate of lime alone. These layers may form complete concentric capsules, or be partial and imperfect. In the latter case the concretion is amenable to the solvent and disintegrating action of the alkaline carbonates; in the former it is wholly beyond the power of such solvents.

Oxalate of lime is insoluble in alkaline carbonates and organic acids; but it dissolves in nitric and muriatic acids. When heated before the blowpipe, it first blackens, and finally leaves a bulky white ash of caustic lime, which blues moistened litmus paper.

During the formation of oxalate of lime calculi, the urine is always acid.

4. *Cystine.*—Gravel and calculi of cystine belong to the rarer species of urinary concretions. They are usually found in the bladder as large calculi, but sometimes they are discharged spontaneously as gravel. I have in my collection a small calculus of pure cystine, weighing only a grain and a-half; it is one of several passed at divers periods by an artisan. It forms a light yellow lenticular mass, with a rough surface. Sometimes vesical calculi of cystine attain a weight of three or four ounces. They are usually egg-shaped, of a full honey-yellow colour, mamillated on the surface, and lustrous, as if studded with minute crystals. When cut into, they show a radiated structure, and an obscurely transparent brilliance like yellow bees'-wax. They are usually composed of pure cystine, unmixed with any other substance. Sometimes they have a nucleus of uric acid. In a specimen in the Museum of the Manchester Infirmary (Fig. 34) the central nodule is uric acid; around this is a body of pure cystine; overlying this a layer of mixed uric acid and cystine; and enveloping the whole a crust of secondary phosphates, mixed with cystine.

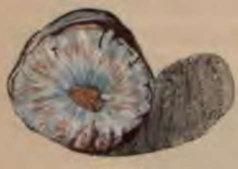

FIG. 34.
Section of a cystine calculus, with a nucleus of uric acid, and an external coat of phosphates.

Cystine calculi possess the curious property of assuming a pale green colour when long exposed to full daylight. The specimen just referred to afforded an interesting example of this change. The calculus had been divided equatorially; one half lay in the cabinet with its cut surface downwards, and the other half with the cut surface upwards, exposed to the light. The latter had a delicate emerald green tint, while the former preserved its original yellow colour.

Another curious circumstance in the history of cystine, is its tendency to run in families. Dr. Marcet gives an account of two brothers in whose kidneys cystine calculi were found. Both Lenoir and Civiale extracted cystine calculi from the bladders of two brothers. Toel relates the history of two sisters who voided cystine with the urine.

Cystine calculi are much more friable than uric acid or oxalate of lime. They are easily scraped with the nail, and offer especially favourable objects for treatment by lithotrity.

Cystine is recognised with great facility. If a particle be placed on a watch-glass, or on a slip of glass, and treated with caustic ammonia, it speedily dissolves; by exposure to the air for a few hours, the volatile alkali exhales, and beautiful six-sided crystals are deposited, which are highly characteristic (see Fig. 12). Cystine is also soluble in the mineral acids; and in the fixed alkalies and their carbonates; but it is precipitated by organic acids and by carbonate of ammonia.

5. *Xanthine* calculi are excessively rare. (See XANTHINE, p. 59.)

6. *Fatty or saponaceous concretions. Urostealith* (of Heller). In the Museum of the College of Surgeons of London, there are two magnificent specimens of vesical calculi, composed of a central fatty or saponaceous mass surrounded with a thick

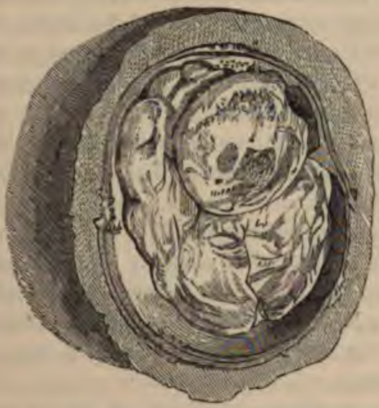

FIG. 35. Section of a fatty or saponaceous concretion (*urostealith*) surrounded with phosphates—from the Museum of the London College of Surgeons.

investment of phosphates (Fig. 35). Both belonged to Hunter's collection, and both are figured and described in the catalogue of calculi published in 1842. They are described as "consisting of the earthy phosphates deposited upon a mass of oleate and margarate of lime." This mass is of a light yellow colour, and its irregularities correspond with those of the cavity in which it loosely lies. At p. 129 of the catalogue, the following ingenious remarks are made respecting the probable origin of these stones :—"On account of some real or supposed disease of the bladder, a solution of soap has been injected into its cavity; mutual decomposition between the soap and the salts of the urine has been the necessary result; the alkali of the former uniting with, and forming soluble compounds with, the phosphoric and other acids of the urine, while the earthy bases of the urine have precipitated, in combination with the fatty acids of the soap, in the form of a semi-gelatinous sparingly soluble compound, being in fact an earthy soap; this substance, acting as a foreign body in the bladder, has induced the deposition of the phosphates, and given rise to the formation of a calculus."

The fatty or saponaceous masses here described are probably of the same nature as those described by Heller in 1845, and named by him *Urostealith*.[*] Only one other case has been published; it was observed by Dr. W. Moore in 1853.[†]

Heller's patient was a man, 24 years of age, who passed a number of small concretions about as large as peas. When fresh, they were soft and elastic, like india-rubber, but dried into hard, brittle, wax-like masses. They dissolved readily in caustic potash, forming a soap. They also dissolved readily in ether, but with difficulty in alcohol. In hot water they did not dissolve, but softened. They melted with heat, and eventually burned with a bright yellow flame, exhaling an odour of shellac and benzoin. They contained a large quantity of earthy phosphates.

Dr. Moore's specimens consisted of two very small dark-brown calculi, which had a soft wax-like consistence, and appeared to consist of a lime soap. They partly dissolved in hot alcohol; and the solution, when cold, deposited a whitish matter, which exhibited numerous fat globules, but no crystalline plates. When incinerated before the blow-pipe they yielded a white, alkaline, calcareous ash. A year later, Dr. Moore received from Dr. R. Adams two calculi taken from the body of this patient. One was a large phosphatic stone, in the centre of which was a cavity containing some of the same dark-brown substance. Dr. W. Davy, who examined a portion of this, judged it to be composed of lime "in combination with the fat or waxy substance forming some organic combinations with the fatty acids."

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[*] Harnconcretionen, p. 146; also Heller's Archiv, Bd. II., p. 1.

[†] Dublin Quarterly Journ. of Med. Science, vol. xvii., p. 473.

7. *Carbonate of lime.*—Concretions of carbonate of lime are of extreme rarity in the human subject. Those which have been described, were of small dimensions, varying from the size of a pea to that of a hazel-nut, smooth on the surface, grey, yellowish, or bronze coloured—sometimes with a metallic lustre, and generally very hard.—(See CARB. LIME, p. 71.)

8. *Basic phosphate of lime or Bone-earth.*—Concretions of this substance alone are very rare. They were formerly confounded with the mixed phosphates which constitute the secondary deposit. They vary in size from a pea to a hen's egg. They are white and chalky in appearance, and of a soft, smoothish exterior, with an earthy fracture. Sometimes their texture is loose, sometimes very compact.

Bone-earth rarely alternates with any other deposit; occasionally, however, it does so. There is a fine specimen in the museum of the Manchester Infirmary, in which bone-earth alternates with uric acid.

When the urine is rendered alkaline by alkalising salts, or becomes alkaline after a meal, the bone-earth phosphate is sometimes abundantly deposited; but, from its uncrystalline condition, it has very little tendency to agglomerate into concretions. Patients may pass an alkaline and turbid urine (from this cause) for months, without practically any risk of the formation of a stone, as is daily witnessed in the now favourite method of treating acute rheumatism by alkalies.

9. *Mixed or secondary phosphates* (*fusible calculus*).—The composition and production of this deposit from ammoniacal urine has been already explained. It rarely forms the entire

of a urinary calculus; but more commonly encrusts calculi of some other species, or an extraneous body which acts as a nucleus (Fig. 36). Concretions of this substance are frequently formed around the inequalities of fungous or other growths of the urinary organs. Calculi of the mixed phosphates may go on increasing for an indefinite period, and completely fill the bladder, attaining a weight of 10, 20, or even 30 ounces.[†]

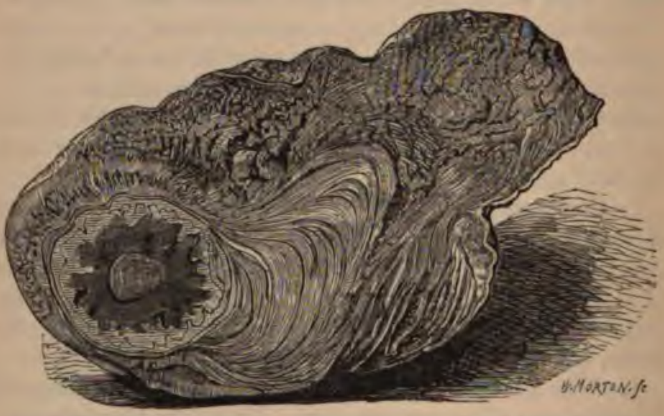

FIG. 36. Section of a concretion, consisting of a vast mass of the mixed phosphates, deposited on a calculus of oxalate of lime.[*]

In their physical characters, fusible calculi most resemble the bone-earth phosphate. They are usually lax and friable, composed of concentric laminæ, or irregular; often studded on the surface with brilliant glistening points of triple phosphate crystals. They readily break down under the lithotrite; but the general irritation of the system, and the frequent co-existence of grave anatomical lesions in the urinary passages or the kidneys, render these cases unfavourable subjects for operation. They are especially suited for a solvent treatment by means of acid injections, thrown into the bladder in the manner practised by Sir B. Brodie.

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[*] From a drawing in the possession of Mr. Southam. The history of this stone (which was successfully removed by the recto-vesical operation) is given by Mr. Southam in the 42nd vol. of the Medico-Chirurgical Transactions.

[†] Dr. Uytterhoeven, of Brussels, withdrew, by the supra-pubic operation, from the bladder of a man, aged thirty-nine, an enormous oval concretion weighing 40½ ounces, and measuring round its longest diameter 17 inches. I believe this is the largest ever extracted from a living person. It had been growing from the age of twelve. (Leroy d'Etiolles (fils), Traité pratique de la Gravelle, Paris, 1863.)

Chemically, this concretion is characterised by fusing into an enamel, when strongly urged by the blow-pipe. It is very soluble in acids, especially the mineral acids; but wholly insoluble in water and alkalies.

10. *Fibrine and blood concretions.*—Marcet gives an account of a small calculus about the size of a large pea, which was passed, after much suffering, by a gentleman between 50 and 55 years of age. He had been suffering for two or three years from symptoms of urinary calculi, and had previously passed three similar concretions. The specimen examined by Marcet had a yellowish-brown colour, somewhat resembling bees'-wax. Its hardness was also nearly that of bees'-wax. Its surface was uneven, but not rough to the touch; it was somewhat elastic. When examined chemically, it answered to reactions of fibrine.

A small concretion, about the size of a small pea, was handed to me for examination by my colleague, Mr. Beever. It had been passed by a man of thirty-five, whose urine was not albuminous. Its texture was hard and brittle, its external surface rough, its colour dark reddish-brown. It swelled into a voluminous coal under the blow-pipe, and, when fully incinerated, left a very scanty white ash. It was evidently composed of inspissated blood.

A patient whom I recently saw with Dr. Holland was in the habit of passing numbers of blood concretions of a softer texture. He had previously suffered from hæmaturia.

Numerous similar concretions were found loose in the infundibula and pelvis of the kidney, in the case of ruptured kidney already described at p. 100.

My collection contains a very fine blood concretion, taken from the bladder of a sheep (Fig. 37). The specimen was presented to me by Mr. Lund. It is as large as a small walnut, very light—weighing only 37 grains. It is nearly spherical, and exceedingly rugged on the surface, which is studded all over with reddish-black warty projections. This dark warty part forms the outer crust of the concretion, is very brittle, and breaks with a lustrous fracture. When sawn through, the rough outside crust is found to be about a line thick; it invests an oval body, which has an even, sharply defined outline. The body has the appearance of baked clay; it is of nut-brown colour, and easily scraped with the nail. It breaks with a dull fracture, like a piece of catechu. Examined chemically and microscopically, both body and crust were found to possess the characters of concrete blood. The scanty ash obtained by calcination gave abundant evidence of iron.

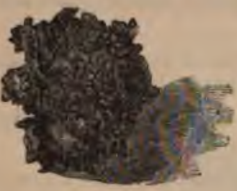

FIG. 37.

Blood concretion from the bladder of a sheep.

All these instances appear to have been connected with the occurrence of renal hæmaturia. Such concretions sometimes serve as nuclei for uric acid or oxalate of lime calculi.*

11. *Prostatic calculi.*—Although these are not, strictly speaking, urinary products, they are, in very rare instances, discharged spontaneously with the urine, and therefore deserve some notice on this connection.

Mr. H. Thompson, who has investigated this subject with great care, states that the existence of concretions in the prostate is almost universal after the age of puberty. He found them invariably present in seventy prostates which he examined from persons above twenty.†

They begin as minute, globular, transparent bodies in the follicles of the gland. At first, they are wholly composed of an albuminous matter, arranged in concentric layers round a vesicular nucleus. But as they grow, they are gradually more and more impregnated with mineral matter, until at length they attain the hardness of the hardest urinary calculi. As a rule, they produce no symptoms, and their existence is, perhaps, hardly to be looked on as a disease. They usually vary in size from a poppy seed to $\frac{1}{2000}$ of an inch.

In some cases, however, the process does not stop here. The earthy material is deposited in great quantity; and large concretions are formed, which encroach on the glandular tissue, and project into the urethra in the form of oblong masses, which require operative procedures for their removal.

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* Wilson's Lectures on the Urinary Organs, p. 181.

† H. Thompson, On the Enlarged Prostate.

A remarkable and, so far as I know, unique example of the spontaneous expulsion of vast numbers of minute prostatic calculi recently fell under my notice.

The patient was a gentleman, seventy years of age, suffering from enlarged prostate, under the care of Mr. George Hunstone, of this town. On the 20th of April, 1864, Mr. Hunstone brought me a specimen of the urine for examination. It was ammoniacal, and contained a good deal of pus. At the bottom of the phial were a large number of minute amber-coloured calculi—the largest of which were about the size of poppy-seeds, and the smallest only just visible to the naked eye, as bright specks. On subsequent occasions Mr. Hunstone brought me additional quantities of urine containing similar bodies. Altogether I obtained about eight grains of these calculi; they were easily separated from the urine by levigation and decantation. Mr. Hunstone stated that the patient had been in the habit of voiding these calculi for some months, at frequent intervals. The patient died some few months back, and, unfortunately, no opportunity was afforded of making a *post-mortem* examination.

The largest of the specimens in my possession is about the size of a mustard seed; there are a good many as large as poppy-seeds; but several hundreds are less than a quarter of this size, and many thousands are still smaller. They are mostly spherical in shape; many are rudely cubical or pyramidal. They possess a full amber colour, and are finely translucent. Under the microscope they present the appearances represented in Fig. 38, and exhibit an infinite series of concentric lines. The centre or nucleus is variously composed. In some of them it is an object resembling a glandular cell, in others a prismatic crystal, in others amorphous earthy-looking matter. In some, again, the nucleus is double, in others, treble, or even quadruple (Fig. 38). With polarized light they display a dark cross, as represented in the lower right-hand corner of the figure. When crushed they break into angular fragments.

The calculi dissolve rapidly in mineral acids, with abundant disengagement of carbonic acid—leaving ragged, brown, flaky, organic remnants. Acetic acid acts very slowly upon them, without disengagement of carbonic acid; but in the course of two or three days all the mineral matter is taken up, and the animal matrix is left, as soft, light balls, preserving the stratified appearance of the original calculi, but with a diminution of their original translucency. They are unaffected by caustic potash. The murexid test yields not the slightest evidence of uric acid. When heated to whiteness before the blow-pipe, their surfaces fuse into a brilliant iron-grey enamel, which protects the deeper parts. As the incandescent calculi cool, the enamelled surfaces crack into numerous minute polygonal spaces. When pulverised and incinerated, the resulting ash does not change the colour of moistened litmus. The solution of the calculi in muriatic acid throws down an abundant, white, flocculent

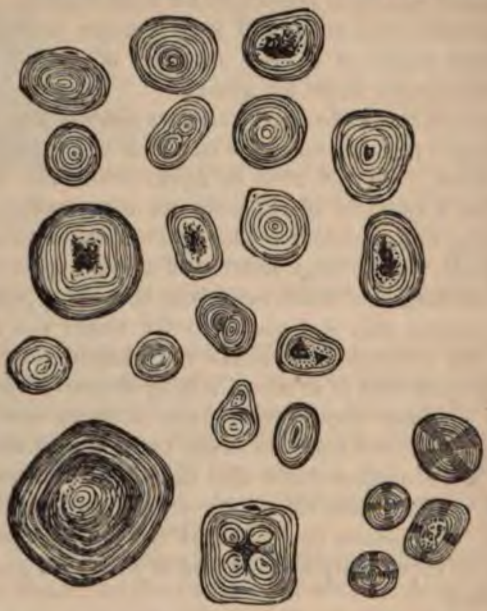

FIG. 38. Prostatic calculi spontaneously voided with the urine—highly magnified.

precipitate, when saturated with caustic ammonia. These reactions indicate that they are composed of an animal matrix impregnated with a mixture of phosphate and carbonate of lime.*

ON THE DIAGNOSIS OF THE SPECIES OF URINARY CALCULI WITHIN THE BLADDER OR KIDNEY.

It would greatly facilitate the choice of the most appropriate treatment, in an individual case of urinary calculus, if it were possible to ascertain beforehand, the exact nature of the concretion. This remark applies equally to surgical and to medical treatment, but more strongly to the latter than to the former.

The degree of precision of this knowledge, attainable in different cases, varies a good deal. The most certain knowledge is gained when a person who has been in the habit of spontaneously voiding small concretions becomes afterwards the subject of stone. In such a case, the examination of the calculi previously passed (supposing them to have been preserved) throws a sure light on the nature of the one retained—provided the epoch at which the former were voided be not too remote, and the characters of the urine continue to correspond.

In the absence of this kind of evidence, certain knowledge of the nature of the stone is rarely attainable; but still, it is generally possible to indicate—from the character of the urine, the constitution of the patient, and the known relative frequency of the several species of stone—*with strong probability*, the species to which it belongs; and also (and with still greater certainty) some of the species to which it does *not* belong.

With regard to the character of the urine, the most important indications are supplied by the nature of its re-action, and the character of the deposit which may be precipitated from it. The re-action of the urine may be (*a*) acid; (*b*) alkaline from fixed alkali; or (*c*) alkaline from carbonate of ammonia.

a. If the urine be *acid*, the stone is almost sure to be uric acid or oxalate of lime, or a mixture of these two.* These two deposits alternate with each other so frequently, and at such short intervals, that, if the urine be free from a uric acid or an oxalate of lime sediment, there is nothing to indicate directly, which of the two species the stone belongs to. But, as uric acid is much more common than oxalate of lime, the probabilities are considerably in favour of the former. If the urine, on cooling, deposit abundantly either uric acid or oxalate of lime, and, *à fortiori*, if either of these deposits are found in the urine at the moment of emission, there is strong probability that the *surface* of the stone is of the same nature; but this gives no warrant of the composition of the deeper strata.

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* I have placed a specimen of these curious calculi in the Museum of the London College of Surgeons.

Vesical calculi are usually more complex in their composition than renal calculi. The latter are almost always composed of one single ingredient; but the former are frequently composed of more than one ingredient. The longer a calculus has resided in the bladder, the more complex will its composition probably be; and conversely, the more recent its descent from the kidney, the more likelihood that it is composed of but a single ingredient. If, therefore, the urine be acid, and the calculus of recent date, the probabilities are greatly increased that it is composed of uric acid alone.

As mulberry calculi have rough surfaces, they usually produce more violent irritation of the bladder than the smoother stones composed of uric acid. This indication is, however, of little practical value, and the exceptions to it are numerous.

Persons of gouty disposition are more likely to be the subjects of uric acid, than of oxalate of lime calculi.

b. If the urine be *alkaline from fixed alkali*, the stone will be composed either of bone-earth phosphate or carbonate of lime. Both are of extreme rarity.

c. If the urine be *alkaline from carbonate of ammonia*, the composition of the nucleus and body of the calculus can no longer be divined; but its surface or crust is sure to be composed of the mixed phosphates. The depth of this crust can only be conjectured from the intensity of the ammoniacal reaction, the quantity of pus and flakes which are discharged with the urine, and the length of time during which this state of urine has persisted. Care must be taken to ascertain if the urine be ammoniacal *at the moment of emission;* for in most cases of stone there is some degree of cystitis; and the presence of pus causes a urine which was passed acid, speedily to become ammoniacal. The degree of the ammoniacal reaction is best judged of by the intensity of the ammoniacal odour, by the gelatinised, or loose, condition of the pus, and by the abundance of triple phosphate crystals. If the urine be only very feebly ammoniacal, or have only recently become ammoniacal, the phosphatic crust may be only a thin film. In the case of large or old phosphatic concretions, fragments of phosphatic *débris* are frequently voided with the urine. If the ammoniacal reaction of the urine is once established in a case of stone, it seldom afterwards gives place to an acid reaction.

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* Concretions of xanthine, fibrine, and fatty matters, are altogether left out of consideration, on account of their extreme rarity. Cystine is also excessively rare; and if cystine crystals be not found in the deposit, it may, practically, be likewise excluded.

MEDICAL TREATMENT OF GRAVEL AND CALCULI.

CHEVALLIER—On the Dissolution of Gravel and Stone in the Bladder, (translated by Edwin Lee). Med. Gaz. 1837, p. 430.

CH. PETIT—Du Traitement Médical des Calculs Urinaires par les Eaux de Vichy. Paris, 1834.

CH. PETIT—Nouvelles Observations de Guerisons, etc. Paris, 1837.

CH. PETIT—Du Mode d'Action des Eaux Minérales de Vichy. Paris, 1850.

CIVIALE—Du Traitement Médical de la Pierre. Paris, 1840.

Two objects are to be held in view in the medical treatment of gravel and calculi, namely, (A) to prevent the formation of a concretion when a tendency thereto exists; and (B) to dissolve or facilitate the expulsion of concretions already formed. The treatment of the organic lesions which are incidental to the presence of calculi in the urinary passages, will be considered—in so far as they implicate the kidneys and their immediate appendages—in Part III. with the other organic affections of the kidneys.

(A) PREVENTIVE TREATMENT.

The disposition to the production of gravel and stone generally passes by undetected, until a concretion is actually formed.

The general health is, usually, not markedly disturbed, and the local symptoms only attract attention when the urinary passages begin to resent the presence of the foreign body.

Sometimes, however, the practitioner becomes aware beforehand, from the character of the urine or other circumstances, that the formation of a stone is a probable event unless preventive means be adopted.

The occurrence of a deposit of uric acid or oxalate of lime after the urine has stood some hours, indicates no special risk of the formation of a stone; but if either of these substances be voided *with* the urine, as gravel, such a risk does certainly exist, and demands to be provided for. Again, if the urine, although clear when voided, lets fall a crystalline deposit *before it has completely cooled*, as may sometimes be seen, especially in children, the danger of the formation of a stone cannot be overlooked. The presence of cystine in the urine is, at all times, a circumstance which demands precautions against the formation of a calculus. The existence of an ammoniacal state of the urine, also, always involves a risk of the deposition of the secondary phosphates.

Independently of the existing state of the urine, evidence of a calculous tendency is sometimes obtained from the antecedents of the patient. If the patient have recently voided a concretion with the urine, or if one have been removed from his bladder by surgical operation, there is reason to apprehend a continuance of the calculous tendency, and the formation of a new concretion.

Under any of these circumstances, preventive measures are demanded. These may be divided into *general* and *special*. The former apply to calculous tendencies of every kind; the latter to threatened formation of some particular species of stone.

Among the *general* indications, the most important is to obviate undue concentration of the urine. This is effected by the systematic use of increased quantities of aqueous drinks. The urine is apt to reach the greatest degree of concentration at hours remote from meal-times (especially during the two or three hours which precede a late dinner), and during the hours of sleep. At these periods the flow of the urine is exceedingly scanty; it is long delayed in the bladder before there is any call for its evacuation; its solid constituents are in excessive proportion to the watery parts, so that the urine resembles a supersaturated saline solution; it is also very acid. Here are united all the conditions most favourable to the separation of some of its less soluble components. Dr. Prout pointed out that the recumbent posture, during sleep, furnished an additional source of apprehension, inasmuch as the urine is no longer aided in its descent by the force of gravity; it therefore lingers and accumulates in the pelvis of the kidney, and is liable to deposit some of its constituents therein.

All these untoward contingencies are obviated by the simple expedient of taking a tumbler of water a couple of hours before dinner, and another before retiring to rest. By this means the urine is diluted, and its escape hastened at the periods when it would otherwise be dangerously saturated, and unduly delayed in the excretory conduits. Two other points are worthy of attention, with a view of maintaining the urine in a state of safe dilution, and providing for its undelayed expulsion. These are: first, that a too great interval shall not elapse between any two meals; and, secondly, that the period devoted to rest in bed shall not be too prolonged. From observations recorded in a previous page, it is seen that a meal both renders the urine more abundant, and lessens its acidity. An individual who shows a tendency to calculous formations should, therefore, be directed to take four or five light meals during the day, at about equal intervals, and to rise betimes in the morning.

When the nature of the calculous tendency is ascertained, either from the character of the deposit, or from the composition of a previously voided concretion, further and *special* precautions should be recommended.

If the tendency be to the precipitation of *uric acid*, the acidity of the urine should be lowered by the moderate employment of the acetate or citrate of potash. A drachm of either salt may be taken in a tumbler of water at bed time, and again on rising in the morning. The diet should be regulated in such manner that animal flesh shall not form a too prominent part of it. Rich wines and heavy meals must be strictly prohibited, and a bland, mostly farinaceous, diet substituted.

The deleterious effects of high diet on uric acid gravel, is aptly illustrated in an example furnished by Magendie :—

> Mr. ——, a merchant in one of the Hanseatic cities, possessed in 1814 an ample fortune, and he lived in accordance with his means—kept a good table, and indulged in its pleasures freely. He was at this time tormented with gout and gravel. Unexpectedly, he lost all his fortune through a political crisis, and was obliged to take refuge in England, where he lived more than a year, almost in poverty, amid numerous privations ; but his gravel completely disappeared. Little by little he succeeded in repairing his affairs ; he resumed his old mode of life, and the gravel was not long in re-appearing. A second reverse robbed him in a short time of all he had gained. He passed into France almost without resources, and his regimen was consonant to his means : the gravel disappeared. Once again his industry restored him to a life of plenty and ease ; he abandoned himself again to the indulgences of the table, and with them appeared once more his old enemy the gravel.*

The special preventive treatment of *cystine* concretions is identical with that of uric acid.

With regard to *oxalate of lime*, the principal indications are, to dilute the urine by abundant regulated potation of aqueous drinks, and to encourage the action of the skin by baths, frictions, the use of flannel clothing, and exercise in the open air. It is important also to guard against the use of certain vegetables which contain large quantities of oxalates and superoxalates in their tissues. The general use of rhubarb tarts in this country in the spring months, and the use of sorrel as salad in France, are probably frequent causes of oxalate of lime concretions. Magendie records two cases in which it appeared highly probable that mulberry calculi had been produced by the daily use of sorrel (l. c. p. 121). Both these articles should be absolutely forbidden.

Mineral and potable waters which are rich in lime, should likewise be avoided.

Heller recommends alkaline substances, on the grounds that oxalate of lime long digested with alkaline carbonates is resolved into a soluble oxalate, and that uric acid is the source of the oxalic acid which appears in the urine. Experiments performed by myself on mulberry calculi, yielded no evidence that the alkaline carbonates exert any solvent action thereon. As to the second point, alkalies do *not* prevent undue formation of uric acid, but merely facilitate its elimination. Nevertheless, I have seen instances in which rendering the urine freely alkaline, caused an oxalate of lime deposit to temporarily disappear from the urine.

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* Magendie, De la Gravelle, p. 25.

Basic phosphate of lime and *carbonate of lime* (unmixed with triple phosphate) are among the rarest forms of urinary calculi. Against them, the proper precautionary measures are, to endeavour to remove the alkalescence of the urine by the exhibition of carbonic acid waters, and to exclude as much as possible all articles of food and drink which are rich in calcareous salts.

The precipitation of the *secondary phosphates* frequently requires precautionary measures to prevent calculous concretions. If severe cystitis follow lithotomy or lithotrity, there is cause to fear a deposition of phosphatic matter upon some fragment left in the bladder, or on a mass of inspissated pus and mucus. Indeed, whenever the urine is highly ammoniacal, the same danger is not remote. To guard against it, the irritation of the bladder should be allayed by appropriate means, and the viscus should be thoroughly washed out, at least twice a week, with water, or with a solution containing a drachm of the commercial muriatic acid to a pint of water.

(B) SOLVENT TREATMENT.

For therapeutical purposes, urinary calculi may be divided into two classes, viz., those which are *soluble in alkalies*, and those which are *soluble in acids*. To the former category belong uric acid, the urates, and cystine: to the latter, phosphatic and mulberry calculi. Those which are soluble in alkalies may (conceivably) be attacked by alkalising the urine by means of certain salts administered by the mouth, or by injecting alkaline solutions into the bladder. Those which are soluble in acids can only be attacked by the latter method, inasmuch as acids cannot be made to pass through the kidneys, save in insignificant proportions.*

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* Various attempts have also been made to apply galvanism to the solution of stones in the bladder. It was ingeniously conceived that the decomposition of a solution of nitrate of potash within the bladder, by a galvanic current,

It will, however, be shown in the sequel, that alkaline substances used in the way of injections, act so feebly on uric acid calculi, that no useful results can be expected from their operation; also that mulberry calculi are unassailable by any solvent method hitherto proposed; so that the solvent treatment of urinary calculi resolves itself, practically, into two lines of action, viz., attacking uric acid calculi (and their congeners), by alkalising the urine by means of medicines administered internally, and phosphatic calculi, by injecting acid solutions into the bladder.

It is a noteworthy fact, that alkaline substances had obtained an extended reputation in the treatment of calculous disorders, long before the composition of urinary calculi had been discovered. In 1739, a remedy of this class—the nostrum of Joanna Stephens—made so great a noise, that Parliament appointed a commission of professional men to inquire into its virtues. The commission reported favourably, and a reward of £5000 was assigned to Miss Stephens for the secret of its composition. The active ingredients in this nostrum were burnt egg-shells and snails, with Alicant soap. As soon as the secret was divulged, soap, soap-ley (solution of caustic potash), and lime-water, were tried in all kinds of calculous cases. The indiscriminate use of the remedies led, as might have been anticipated, to contradictory results. Both successes and failures were published in large numbers;* and opinion was much divided as to their utility. About this time the successes of Cheselden gave a great impulse to lithotomy, and the use of solvents gradually fell into discredit. The subject was resuscitated in France about a century later, under the inspiration of the great advances then made in chemical science, and especially of the discoveries of Wollaston and Fourcroy into the nature and composition of urinary calculi. The virtues of the alkaline bi-carbonates—and more particularly of the bi-carbo-

would set free, simultaneously, both nitric acid and caustic potash—one or other of which is capable of acting on every variety of stone. But the mechanical difficulties of this proceeding have hitherto proved insurmountable; and the slow action which my experiments prove solutions of caustic potash to have on uric acid calculi, and solutions of nitric acid on mulberry calculi, render it hopeless ever to obtain results of practical utility by this method. See also Heller's Harnconcretionen, p. 99.

* Ploucquet gives a list of more than forty papers and pamphlets, published on the subject about 1740.

nate of soda, the active ingredient of the Vichy springs—were brought into prominence; and a considerable number of cases, successfully treated by these means, were published by Chevallier and Ch. Petit. But again, the absurd claim of universal efficacy brought the solvent treatment into contempt; and for the last twenty years, and more, urinary calculi have been almost wholly abandoned to the surgeon.

A perusal of the literature of these two periods, however, strongly suggested the desirability of subjecting the question to a new examination, with a view of ascertaining the causes of the discrepant experience of past times; and also of indicating with some approach to certainty, what may be rationally expected from a solvent treatment, in what cases it is applicable, and the precise mode of carrying it out effectually.

For the purpose of clearing up these questions, the present writer undertook an extensive series of experiments, and made numerous clinical observations. The facts observed are embodied in a paper read before the Medical and Chirurgical Society on March 28, 1865. To this paper the reader is referred for fuller details. The results obtained seem to demand a considerable modification of the prevailing opinion regarding the inutility of the solvent treatment. They do not by any means indicate the *general* possibility of substituting a solvent for a mechanical treatment of *vesical* calculi; but they suggest an essential improvement in the treatment of *renal* calculi; they also indicate that uric acid and cystine, under certain circumstances, are capable of solution in the bladder, by means of alkaline salts administered by the month, at a rate which admits of practical application; and that, in certain picked cases of this class, a solvent treatment deserves to be resolutely tried, before having recourse to the more dangerous methods of lithotomy and lithotrity.

Attention was naturally directed in the first instance, and chiefly, to uric acid, both on account of its being by far the most common constituent of urinary calculi, and also on account of its offering the greatest probabilities of success. But the inquiry was not altogether confined to uric acid: experiments were also made on the solubility of cystine, oxalate of lime, and phosphatic calculi.

ON THE SOLVENT TREATMENT OF URIC ACID BY THE ADMINISTRATION OF ALKALINE SALTS BY THE MOUTH.

The inquiry respecting uric acid, set out from two known data, viz.:—

First.—That solutions of the alkaline carbonates exercise a solvent action on uric acid.

Second.—That the urine can be rendered alkaline from alkaline carbonates by the administration of certain salts by the mouth.

Starting from these data, a number of preliminary questions immediately presented themselves, which it was necessary to answer before proceeding to the more practical part of the inquiry. These were:—1. Whether is carbonate of potash or carbonate of soda* the better solvent for uric acid? 2. What is the best strength of solution to employ? 3. What is the effect of varying quantities of the solution on the results obtained?

Answers to these questions were sought by placing sections of uric acid calculi, usually weighing about 100 grains, in 10oz. phials, and causing currents of the different solutions, at blood heat, to pass over them at a regulated rate.

1. With regard to the COMPARATIVE SOLVENT POWERS OF CARBONATE OF POTASH AND CARBONATE OF SODA, the experiments indicated clearly that potash dissolved uric acid more rapidly than soda. A solution of carb. potash, containing 30 grains to the pint, dissolved daily, 11·9 per cent. of a uric acid calculus; while a solution of carb. soda of equal strength, dissolved only 10·3 per cent. The potash salt possessed a further advantage in its wider range of solvent power with the stronger solutions. This latter point will be better understood after the effects of solutions of different strength have been considered in the next paragraph.†

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* The experiments were principally directed to ascertain the effects of the alkaline *carbonates*, because all salts which have the power of alkalising the urine to a useful degree, appear in the urine as carbonates. A number of other salts were however tried, viz., neutral and alkaline borates, phosphates, and soaps.

† Several experiments were also made with carbonate of lithia, which has

2. The Strength of the Solution was found to affect its solvent capacity more than any other condition. It soon became apparent that only very weak solutions could yield any useful results. The greatest solvent power was found to reside, in solutions containing from 40 to 60 grains of carbonate to the imperial pint. Below this strength, the power of the solutions gradually declined, until, with solutions containing less than three grains to the pint, the solvent power scarcely exceeded that of ordinary water. On the other hand, with solutions above the strength of 60 grains to the pint, dissolution was impeded, and finally arrested, by the formation of a white crust or coat of alkaline bi-urate on the surface of the calculus. With a solution of 80 grains to the pint, this bi-urate crust was loose and easily detached, like a layer of whitewash; but with a solution of 120 grains to the pint, the crust was tenaceous and adherent, and very little dissolution took place with carbonate of potash, and none at all with carbonate of soda. With solutions of 160 and 240 grains to the pint, there was no loss of weight with potash or soda; the fragments became invested with a thin tough coating of white bi-urate, resembling white paint, which put a stop to all solvent action.*

The following table exhibits the results obtained with solutions of carbonate of potash of varying strength :—

Strength of solution.	Daily average loss of weight.
240 grains per pint	0 per cent.
160 „	0 „
120 „	3·0 „
80 „	9·8 „
60 „	20·2 „
40 „	15·6 „
30 „	11·9 „
20 „	11·0 „
10 „	6·5 „
5 „	6·0 „
2½ „	2·8 „
1 „	1·2 „

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been much vaunted in recent times as a solvent for uric acid. Its power was, however, found much inferior to that of the carbonates of potash and soda. Its reputation seems to have arisen from its comparative insolubility. Only weak solutions of carb. lithia *could* be employed, and these were compared with solutions of potash and soda which were too strong.

* For further information regarding this white coating of bi-urate, see an abstract of a paper by the author in the Trans. of the Brit. Assoc. for the Adv. of Science for 1861; also Beale's Archives for 1862.

The quantity of solution permitted to flow over the stone was, generally, six pints in the twenty-four hours.

3. It was at first supposed that the QUANTITY OF THE SOLUTION permitted to flow over the stone, would greatly influence the rate of dissolution; but on actual trial, the effect of quantity, within the limits necessarily imposed by the capacity of the kidneys to eliminate fluids, proved to be comparatively unimportant. In order to obtain comparable results, different quantities of a solution of uniform strength were passed over the same stone on successive days.

A solution of carbonate of potash, containing thirty grains to the pint, gave the following results:—

Daily flow.	Daily loss of weight.
15 pints	13·0 per cent.
8 „	15·0 „
6 „	10·2 „
4 „	9·5 „

A flow of even one or two pints per day, with a solution of suitable strength, produced a copious dissolution. Two pints of a solution of carbonate of potash, containing forty grains to the pint, caused a daily dissolution of 17·1 per cent.

4. With regard to the ABSOLUTE RATE OF DISSOLUTION attainable, the experiments opened out an inviting prospect. The solutions of maximum solvent power dissolved from ten to twenty per cent. of the calculi in the course of twenty-four hours. If results approaching these could be obtained in the living body, a little consideration will show, that such an impression could be made on a uric acid concretion, in a few weeks or months, as would either entirely dissolve it, or reduce its dimensions to a point, which would enable it to escape spontaneously by the natural passages.

Having disposed of these preliminary inquiries, the next points to be ascertained were: the best way of alkalising the urine, so as to impart to it an alkalescence corresponding to that of solutions of carbonate of potash of maximum solvent power: also to examine the actual effect of alkalised urine passed over uric acid calculi, in a phial, at blood heat.

5. The most convenient WAY OF ALKALISING THE URINE,

was found to consist, in giving frequently repeated doses of the acetate or the citrate of potash. Both these salts are extremely soluble; they are well borne by the stomach; they do not interfere with digestion nor occasion purging. Weight for weight, the two salts were found to possess nearly equal alkalising powers. With some individuals the acetate agreed better than the citrate: with others the converse was the case.

In order to maintain the urine at a degree of alkalescence that should correspond to the maximum solvent power of solutions of carbonate of potash (*i. e.*, an alkalescence equal to about 50 grains of carbonate to the pint), it was found necessary, in adults, to administer from 40 to 50 grains of the acetate or citrate of potash, dissolved in three or four ounces of water, every three hours.

It was found quite impossible to maintain the urine at an *absolutely constant* degree of alkalescence, however short the intervals at which the dose was repeated. The activity of the kidneys oscillates from hour to hour; at one time the urine is secreted abundantly and dilute, and then the degree of alkalescence necessarily falls; at another time it is secreted more scantily and more concentrated, and then the degree of alkalescence rises. When, however, the above dose was exhibited with regularity, every second or third hour, the oscillations rarely passed an alkalescence equivalent to 20 grains to the pint, on the one hand, and 80 grains to the pint on the other: and, as a rule, the alkalescence ranged between 35 and 60 grains to the pint—which corresponds, sufficiently exactly, with the maximum solvent power of a solution of carbonate of potash in water.

6. When urine, alkalised by the internal administration of these salts, was passed over the surface of uric acid calculi, at blood heat, the calculi were found to undergo solution at THE MEAN RATE of 12½ grains in the 24 hours.

In performing this experiment it was found, that, unless the calculus and the phial were frequently cleansed by immersion in water, the urine became ammoniacal, and the calculus became covered over with a crust of the mixed phosphates, which speedily put a stop to the solvent action of the alkalised urine.

An important practical deduction flowed from this fact, viz., *that when an ammoniacal state is developed, the solvent power of alkalised urine is entirely nullified, by the deposition of the mixed phosphates on the surface of the calculus.*

The urine of patients taking full doses of the citrate or acetate of potash, is generally clear, and shows no tendency to deposit, even on standing. But this is not invariably the case ; it is sometimes turbid from deposition of the amorphous phosphate of lime. Two conditions seemed especially to favour this deposition, namely, the febrile state, and the digestion of a heavy meal. The amorphous phosphate is not unfrequently deposited, as we have already seen (p. 24), after a meal, in healthy persons who are not taking any alkalising medicines : the circumstance is, therefore, not to be regarded as an unnatural or hazardous one. It is, further, to be borne in mind, that the amorphous phosphate differs essentially from the mixed phosphates thrown down in an ammoniacal urine. The former is a loose flocculent substance, which shows no tendency to aggregate into concretions ; the latter, on the other hand, is partly crystalline, and speedily encrusts any object brought into contact with it. The establishment of this distinction disposes of one objection which has been urged against alkaline solvents.

It now remains, to bring forward illustrations of the application of the solvent treatment in practice ; to distinguish the cases in which the treatment is applicable ; to lay down directions for carrying it out effectually ; and, finally, to examine some of the objections which have been urged against its employment.

7. ILLUSTRATIONS of the practical employment of alkaline solvents, may be divided into cases of *renal* calculi, and cases of *vesical* calculi.

One of the first rational attempts to treat *renal* gravel of uric acid by alkaline solvents, was made by the celebrated Mascagni in his own person. He gives the following account of his case in the Memoirs of the Italian Society for 1804 :—

> I had been subject for several years to pains in the lumbar regions, and I voided from time to time gravelly concretions of a yellow-ochre and brick-red colour. Knowing that gaseous alkaline fluids had been used in such

cases, I took some on several occasions with benefit. I imagined I could obtain greater effects with carbonate of potash.

In the month of August and September, 1799, having been obliged to lead a sedentary life, I was cruelly attacked with pains in the kidneys, and I voided a considerable number of small concretions, some of which were large enough to be regarded as veritable calculi. They were reddish and crystalline; they were deposited at the bottom of the vessel each time I made water, and I could see their glistening facets through the transparent urine. I was also subject to an excess of acid in the stomach, which was perceived in the mouth. I examined my urine and found in it a free acid, which, as well as the concretions, I recognised as consisting of uric acid.

Having thus assured myself of the nature of the concretions I was voiding, I resolved to make use of the carbonate of potash and to observe the result. I took the first day about a drachm, one half in the morning fasting and the other half in the evening. I dined at one o'clock in the afternoon. This salt dissolved in ten ounces of water had very little taste, it caused no disturbance of the stomach or bowels; but as soon as I swallowed it, it occasioned a considerable disengagement of carbonic acid gas, which was felt in the mouth and discharged by the anus.

The second day I took two drachms, and the third day three drachms; and I continued this dose, dissolved in twenty ounces of water, for ten days. Before using the carbonate my urine was very acid, and intensely reddened blue litmus paper. On the second day the paper changed colour very little, and none at all on the third day. The acid of my urine was therefore saturated. At this epoch the renal pains diminished, and I voided no more gravel with the urine. Afterwards the pains ceased entirely, the urine became less loaded, and *I recognised the potash in excess.*

I ceased to use the carbonate of potash, and for some months I voided no concretions. Being subsequently attacked with the same symptoms, I had recourse to the same remedy, and I obtained the same good effects. I have repeated this medico-chemical experiment every time I have felt the same inconvenience, and always with success. Two years have now elapsed since I voided any concretions, though I no longer make use of the potash.*

The following example from my own practice is not dissimilar :—

In July, 1860, a stout middle-aged gentleman brought to me eleven small concretions, varying from the size of a pea to that of a large pin's head. He had voided these with the urine a few days previously; they were composed of uric acid. He stated that three years before, he was attacked with renal colic, which subsided on the third day with the discharge of a small calculus by the urethra. From this period to the time of my seeing him, attacks of renal colic, terminating in the discharge of small brownish concretions, recurred with great regularity at intervals of three or four months.

The urine was found to be acid and high-coloured : the general health was somewhat impaired by his periodical sufferings.

In projecting the plan of treatment, it was considered that the patient had in all probability a number of similar concretions still lodged in his kidneys. The dissolution of these was the first object; the next was to prevent their formation in the future. Seeing the small size of the concretions, it was thought that by keeping up a persistently alkaline state of the urine for a week or two, complete dissolution of them would be effected. With this view citrate of potash, in two-scruple doses, dissolved in half a pint of water, was administered every three hours for a fortnight. Afterwards the patient took a drachm of the same salt in a tumbler of water night and morning for a period of three months. As no recurrence of the renal pains took place, nor the discharge of any concretions, the medicine was discontinued; but the patient was instructed to take every night before going to bed a tumbler and a half of water. This practice he has continued up to the present time (October, 1864). There has been no return of the symptoms.

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* Magendie, De la Gravelle, p. 85.

A considerable number of examples of the successful treatment of *vesical* calculi by alkaline solvents lie buried in the forgotten publications which appeared in this country about the middle of the last century, when the remedy of Miss Stephens made so great a noise. Some fifteen or twenty cases were also collected by Chevallier and Petit at a later epoch, when the question was resuscitated in France thirty years ago.

Most of these reports are vitiated by the absence of information as to the nature of the stone and the condition of the urine. At the former epoch (1740), urinary calculi were all supposed to be of the same nature, and that an unknown one. At the latter epoch the chemical composition of urinary calculi was indeed known, but some of the most important points in their development were misunderstood; urinary chemistry was still in its infancy; and the same absurd pretension of universal efficacy was put forth on behalf of alkaline substances which swamped their reputation in 1740.

One of the best illustrations from the earlier records is supplied by Dr. James Jurin, who was himself the sufferer.

He was for many years subject to red gravel. At Christmas, 1740, he voided a small stone, after suffering four days from nephritic colic. In January and February following, he perceived unmistakeable symptoms of stone in the bladder. These he describes at great length, and with remarkable clearness.

In March, he began to take lixivium of soap or soap-ley (a strong solution of caustic potash), in gradually-increasing doses, until he reached the amount of an ounce or an ounce and a quarter daily. He took for a single dose one or two tea-spoonfuls of the lixivium diluted with three quarters of a pint of water. The soap-ley which he employed was "one-fifth part heavier than river water" (*i.e.*, its specific gravity was 1200, which is about three times as strong as the liq. potassæ of the London Pharmacopœia).

He continued this treatment for five months. On the 10th of July he voided a small smooth stone of the size of an oat, and of a reddish colour. On the 27th of the same month he voided a second stone. On August the 6th he voided a third stone; and about the beginning of September a fourth. All his symptoms now disappeared, and he discontinued the medicine; but in December he had a return of the vesical symptoms; he also noticed that his urine again furred the chamber-pot, and that he voided a little red gravel, as he had formerly done. He went back to the soap-ley, and in the course of a week parted with a small rough reddish stone. From that time he continued perfectly easy. He still took a couple of tea-spoonfuls of the lixivium each day, and this he found sufficient to keep the urine from furring the utensil.*

The calculi in this case were undoubtedly uric acid, as may be learnt not only from their red colour, but also from an experiment which Dr. Jurin made: he found that they dissolved in the alkaline ley and in lime-water.

Of the cases collected in France, I will only cite one. In Chevallier's essay, ten cases of the successful use of the bicarbonate of soda are recorded. Dr. Petit has contributed some half-a-dozen additional cases illustrating the effects of Vichy waters (which contain 44 grains of bicarbonate of soda to the pint.)

M. de L——, fifty-one years of age, was sounded by Leroy d'Etiolles, who found a stone in the bladder. This he believed to be not large, and suitable for crushing. The patient, however, went to Vichy, and drank the first day seven or eight glasses of the waters. The next day he took fifteen glasses, and the urine, which was previously very acid, became constantly and strongly alkaline. In a few days he took twenty-two and twenty-four glasses. The symptoms, which were before severe, now subsided more and more, and after seventeen days of treatment he voided a smooth uric-acid concretion which bore evident traces of dissolution. From this moment he continued wholly free from symptoms, and was able to take violent equestrian exercise without the least inconvenience.†

The causes which led to the discredit and final abandonment of the alkaline treatment, in spite of the large mass of evidence in its favour, are now easy to understand. The most important of these was the erroneous claim to universal applicability set up for it by its advocates. My experiments prove unequivocally that it is wholly powerless in all cases where the urine is ammoniacal; also in all cases of oxalate of lime calculi, and in every variety of phosphatic calculi. No benefit can be derived from it except in cases of uric acid and cystine calculi, and in these, only where the urine has *not* become ammoniacal. The indiscriminate use of the treatment, therefore, could only result in disappointment. Further, the treatment was carried out in a very imperfect manner. In the earlier period (1740), alkaline substances were given in the form of soap, calcined egg-shells, lime-water, or solutions of caustic potash—all of them nauseous to the taste, apt to derange the stomach, and difficult to administer in sufficient doses to prove efficacious. In the later period (1840), Vichy waters were chiefly relied on. These contain soda, which, as we have seen, is an inferior solvent to potash; and the great dilution of the remedy in the Vichy waters must seriously impair its power.

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* The record of this case is bound up with Rutty's Observations on Joanna Stephens's Medicine for the Stone. Lond. 1742. Another good case is related by Whytt, in his Essay on Lime-water. Edin. 1752: and a third, in which the successful result is vouched for by a *post-mortem* examination made seventeen years afterwards, is recorded in the Philosophical Transactions for 1745, by Dr. Pringle.

† Dr. Ch. Petit, Du Mode d'Action des Eaux Minérales de Vichy, p. 272.

My own experience of the alkaline treatment in vesical calculi, was gathered before some important points were understood, which later inquiries have made clear to me.

My *first* case was one of uric-acid calculus, and in every way suitable for the solvent treatment; but it was carried out very imperfectly, and was not persevered-in sufficiently long to effect complete dissolution. The patient was a boy, four years of age, admitted into the Manchester Infirmary, Dec. 1, 1858. The urine was acid, but did not deposit any crystalline sediment. He was placed under the influence of the tartrate of potash and soda (Rochelle salt), in the doses, at first, of twenty grains, and afterwards of thirty grains, dissolved in from four to six ounces of water, every two hours. The treatment was continued for six weeks. The urine was thereby rendered very feebly alkaline. At the end of this period the sound still disclosed the presence of a stone, and the operation of lithotomy was accordingly performed by my colleague, Mr. Southam, with perfect success. Two calculi were extracted, which together only weighed twenty-two grains; they were composed of pure uric acid, and their surfaces were perfectly smooth, and polished like river pebbles, without a particle of phosphatic incrustation.

My present experience enables me to point out two errors in the plan of treatment followed in this case. In the first place, the quantity of fluid in which the salt was dissolved was much too large; and in the second place, the salt used had too feeble an alkalising power. Rochelle salt, on account of its large proportion of water of crystallisation, has less alkalising power by more than one-third than an equal weight of the citrate or acetate of potash.

Notwithstanding these drawbacks, it is not possible but that a considerable amount of dissolution had taken place. The urine was kept constantly, though feebly, alkaline for six weeks; there was no carbonate of ammonia developed in it, and no trace of phosphatic deposit on the stones. These are conditions in which, as my experiments prove, uric acid *must* undergo solution. The two calculi when extracted weighed only 22 grains; and yet one or both of them must have existed in the bladder for a period of three years, for the symptoms of vesical calculus had been distinctly noticed for so long. It is scarcely conceivable that these stones had not attained, in this length of time, a greater magnitude than they possessed when extracted: and it seems not too much to suppose that had the treatment, imperfect though it was, been persevered-in for another week or fortnight, the size of the concretions would have been sufficiently reduced to permit their escape spontaneously by the urethra.

My *second* case was a boy, aged twelve, an inmate of the Manchester Children's Hospital, under the charge of Dr. Borchardt and Mr. Smart, who kindly permitted me to direct the treatment. The urine was acid; it contained a little pus, and had an inordinate tendency to deposit uric-acid crystals.

On Sept. 19, 1860, the patient was directed to take twenty grains of the acetate of potash in two ounces of water every three hours. This treatment was continued for thirty-four days; the urine was rendered thereby continuously alkaline. At the end of thirty-four days, the stone, being still found on sounding, was successfully extracted by Mr. Smart.

The calculus weighed 180 grains, and its form was a flattened oval; it was found to be composed of alternating layers of uric acid and oxalate of lime; and its surface presented a most peculiar appearance, which furnished an interesting and irrefragable proof of the solvent action of the alkalised urine on the uric-acid layers of the stone.

The outermost layer consisted of uric acid, and over the larger circumference of the stone it had a thickness in its deepest parts of about a line and a-half; but on the flattened surfaces the uric acid was dissolved away, and the subjacent layer of oxalate of lime cropped through it to a considerable extent. On one side the exposed patch of oxalate was as large as a sixpence, and presented the ordinary tuberculated appearance and dark brown colour of a mulberry calculus. On the opposite side, two islets of oxalate were uncovered, each about the size of a large split-pea. Surrounding the exposed patches of oxalate were found the remnants of a thinner, more superficial, and incomplete layer of oxalate of lime. The irregular patches of this latter layer occupied a higher level than the surrounding surface of uric acid; and here and there little elevations of uric acid could be seen surmounted with a shield of oxalate of lime. These elevations were partially undermined; the uric acid had been attacked by the solvent, and the protecting shield of oxalate of lime was in process of being thrown off by the gradual melting of its support.

The general surface of uric acid had a characteristic water-worn appearance. There were no minute mamillations such as usually stud the surface of uric-acid concretions; but the surface was undulating, and the hollows and intervening ridges were perfectly smooth. No trace of phosphatic deposit existed on any portion of the stone.

Complete solution of the calculus was not possible in this case. A concretion composed of a uniform mixture of uric acid and oxalate of lime, was found to be attacked with considerable facility, by a solution of carbonate of potash, in the phial; and the present specimen shows that thin and incomplete layers of oxalate of lime may be undermined and disintegrated by alkalised urine; but if the stratum of oxalate be complete, and entirely invest the stone, it puts an absolute bar to further solvent action. This was the case in the instance before us. The partially uncovered layer of oxalate of lime surrounded the entire stone; and as soon as the dissolution of the superincumbent layer of uric acid had been completed, no further diminution of size could have taken place.

The treatment was not carried out in this case as efficiently as it might have been. The dose of the acetate should have been nearly double: this would have considerably more than doubled its solvent effect. The alkalescence of urine produced in a boy of twelve by twenty grains of the acetate every three hours is but feeble, and does not approach the highest solvent power capable of being imparted to the urine.

My *third* case was a boy of six, admitted under my care into the Manchester Infirmary, on Jan. 27, 1862. The urine was acid, and singularly free from pus, blood, and other organic elements. He was placed under the influence of citrate of potash—at first, in the dose of twenty grains in six ounces of water every two hours. This was speedily raised to twenty-five grains, and continued with great regularity for two months. At the end of this period the dose was raised to thirty grains, given two-hourly in six ounces of water, and continued for a month longer. At the end of the third month the stone was still felt on sounding. The patient was then transferred to the care of my colleague, Mr. Southam, who successfully extracted the stone by the lateral operation. It proved to be a fine specimen of mulberry calculus, excessively rough on the surface, and not bearing the slightest traces of dissolution. Not a particle of phosphate existed on its surface. When sawn across a nucleus of uric acid was displayed. The outer crust of oxalate of lime was about a line and a quarter thick.

The solvent treatment was carried out with undoubted efficiency in this case, for the space of three months; but, of course, wholly in vain, owing to the impenetrable layer of oxalate of lime with which the stone was invested. The only defects which my later experiments enable me to point out, were the unnecessarily large amount of liquid administered and the unnecessarily frequent repetition of the dose. If the same dose had been given in half the quantity of water, and repeated every third hour, an equal effect on the urine would have been produced.

These three observations permit a deduction of great importance to be drawn from them, namely, *that a continuously alkaline state of the urine does not determine any precipitation of the earthy phosphate on the stone, so long as the urine is free from ammoniacal decomposition.*

8. DISCRIMINATION OF THE CASES IN WHICH THE SOLVENT TREATMENT IS, AND IS NOT, APPLICABLE. The first and most general limitation is :—

A. *The solvent treatment is inapplicable to all cases in which the urine is alkaline.*—The loss of the acid reaction of the urine in calculous cases, is due, in the overwhelming majority of cases, to ammoniacal decomposition from vesical catarrh. This state of the urine determines the precipitation of a phosphatic crust on the surface of the stone, and withdraws it completely from the influence of alkaline solvents.

B. *When the urine is acid*, the case may be regarded *prima facie* as suitable to the solvent treatment; but there are still numerous limitations which reduce the cases really suitable within a much narrower compass.

(*a*.) In the first place, *all those cases are excluded in which it is known or strongly suspected that the stone is composed of oxalate of lime.* This is sometimes ascertained from the patient having previously voided concretions of oxalate of lime; sometimes the character of the urine yields indications of the nature of the stone; if it deposit on cooling an abundant sediment of octahedra, or dumb-bells, the strong inference is that the stone is composed wholly or in part of oxalate of lime.

(*b.*) When the examination of the urine and the previous history of the patient give *no indication of the nature of the stone*, we are left in doubt (supposing the urine to be acid) whether it is composed of oxalate of lime or uric acid, or of alternating layers of these two substances. There are no data at hand to form an opinion as to the probabilities here involved. Different countries, and even different districts of the same country, show considerable diversities in the relative proportion of uric-acid and mulberry calculi. Renal calculi also differ essentially in regard to this point from vesical calculi. The former are generally composed of a single substance: and in about five-sixths of the cases this is uric acid. The latter, if they have sojourned any considerable time in the bladder, are frequently composed of two or more substances arranged in alternate layers (see p. 228).

In cases of *renal calculi* the patient should evidently have the benefit of the doubt. No other treatment than that by alkaline solvents, is open to the choice of the practitioner; and if the calculi should be composed of oxalate of lime, the alkaline treatment will not aggravate, if it do not ameliorate, the state of the patient.

In cases of *vesical calculi* the question stands differently. The solvent treatment comes here into competition with the mechanical methods of lithotomy and lithotrity, which long experience have stamped with success. It is no longer a question of the mere possibility of removing a calculus by means of solvents, but of doing it with less risk than by lithotomy or lithotrity.

Future experience can alone decide, whether it is better, in cases of this class (where the nature of the stone is quite uncertain), to consign them at once to the operating table, or to give a preliminary trial to the solvent treatment. It would appear from the cases reported in the preceding pages, that patients who have undergone such a trial may be afterwards transferred to the surgeon with undiminished chances of a successful operation. Probably the most advantageous course to follow, if the stone be a small one, would be, to try the solvent treatment for a limited period—for six weeks or two months—and if unsuccessful at the end of that time, to proceed without further delay to operation.

(*c.*) *When the stone is known to be a large one*, the solvent treatment should not be attempted. The presence of a large stone in the bladder is itself a perpetual source of danger; and the larger the stone, the greater the probability that it contains one or more layers of oxalate of lime, which will resist the solvent. The length of time which a stone above the weight of an ounce would require for dissolution, also detracts greatly from the advantages of the solvent treatment, as compared with the swifter, though less safe, method of lithotomy.

(*d.*) The cases of vesical calculi *which are especially suitable for the solvent treatment, are those in which it is known or strongly suspected that the concretion consists of uric acid, and has not yet attained any great size.* It not unfrequently comes to pass that an individual who has previously, at divers times, spontaneously voided small uric-acid calculi, becomes afterwards the subject of vesical calculus. If such a case come under treatment soon after the first appearance of symptoms of stone in the bladder, it is one peculiarly promising for the solvent treatment. The stone is sure to be small, and it is almost certain to be wholly composed of uric acid. A dissolution of twenty or thirty grains would reduce the stone sufficiently to enable it to traverse the urethra. A more rational, safe, and certain plan of treatment is scarcely conceivable in any disease.

(*e.*) It is probable that the solvent treatment, judiciously carried out, will prove a useful *adjunct to lithotrity*. It is, however, essential to its employment that no vesical catarrh, with ammoniacal decomposition of the urine, ensue after the operation. If the urine maintain its acidity after the stone is crushed, and if the fragments discharged prove to be uric acid, then the solvent treatment might be expected to act advantageously by obviating the inconvenience and danger of repeated sittings.

To sum up in the affirmative:—the solvent treatment is only applicable in those cases of vesical calculi in which *the urine is acid; the stone not large; its composition known to be uric acid or strongly suspected to be such.*

9. RULES FOR CARRYING OUT THE SOLVENT TREATMENT.—The action of alkalised urine is essentially slow; quick solution, by any manner of applying it, is impossible. To make up for this defect, its operation must be continuous and incessant. To rest content with alkalising the urine for a few hours each day, is not only to reduce the solvent effect to an insignificant quantity, but, sometimes at least, to nullify it altogether. I have known urine kept continuously alkaline by acetate of potash for many successive days, recover its acidity and deposit uric acid within a few hours of the latest dose. It is also of great importance not only to keep the urine continuously alkaline, *but to keep it alkaline to a certain degree.* The experiments described at p. 237 prove that solutions with an alkalescence below three grains of carbonate of potash to the pint, have scarcely a greater effect on uric-acid calculi than simple water. A feebly alkalised urine acts so slowly, that (in cases of vesical calculi) the delay incurred counterbalances the safety of the treatment as compared with mechanical means, and robs it of the preference which it might otherwise deserve.

To secure a continuous alkalescence, the dose should be repeated at intervals of not less than three hours, and it should be given with rigorous regularity during the waking hours. A dose should be taken the last thing before retiring to rest, and another in the course of the night. Of course a patient should not be disturbed from sleep in order to take a dose of medicine; but patients with vesical calculi, scarcely ever are able to pass the night, without awaking spontaneously once or more to empty the bladder.

The best salts for administration are the acetate and citrate of potash. Of the former, the dose for an adult should be from 40 to 60 grains dissolved in 3 or 4 ounces of water; for children, the dose should range from 20 to 30 grains. The citrate (anhydrous) has nearly the same alkalising power as the acetate. The citrate of potash of the shops is of uncertain strength, and often exceedingly impure. The best plan is to prepare the solution directly from the crystallised bicarbonate of potash and crystallised citric acid. The bicarbonate, when saturated with citric acid, forms almost exactly its own weight of anhydrous citrate; so that when 40 grains of bicarbonate of potash are saturated with the proper quantity of citric acid, there result 40 grains of citrate of potash.

The following prescription yields a solution containing one drachm of the citrate in each fluid ounce :—

℞ Potass. bicarb. ʒxii.
Acid. citric. ʒviii., gr. xxiv.
Aquæ, ad ℥xii.

The dose of such a solution for an adult, is 6 or 8 fluid drachms mixed with 3 or 4 ounces of water ; and for children, 3 to 6 fluid drachms diluted in the same proportion.

In conducting the treatment, it is essential that the freshly-voided urine should be frequently examined. If at any time it show signs of ammonical decomposition the treatment should be suspended. The advent of this state is indicated by the offensive ammoniacal smell of the urine and the increase of pus and flaky matter in it. As long as the urine continues sweet *when voided*, no fear need be entertained of the deposition of the mixed phosphates on the surface of the stone.

10. THE OBJECTIONS urged against the alkaline treatment have been chiefly three :—

(*a*.) It has been alleged, that by rendering the urine alkaline, there is danger of the precipitation of the phosphates on the surface of the stone. The facts advanced in the preceding pages dispose of this objection completely. If there be ammoniacal decomposition of the urine, the phosphates are deposited whether alkaline medicines be given or not, and the concretion goes on increasing ; but if the urine be alkaline solely from fixed alkali, not a particle of phosphatic deposit takes place.*

(*b*.) It has been said that the natural reaction of the urine is acid ; and therefore, that to render it alkaline is to introduce an unnatural state, which cannot fail to act deleteriously on the general health. In a state of fasting the natural urine is doubtless always acid ; but the researches of Dr. Bence Jones, fully confirmed by my own (see p. 23), show that the urine is normally alkaline (from fixed alkali) for several hours daily, after meals, in many, if not all, healthy persons. So that the maintenance of an alkaline reaction of the urine by fixed alkali is by no means so unnatural a state as some have supposed.

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* A want of knowledge of the essential difference between urine alkaline from fixed alkali and urine alkaline from carbonate of ammonia, runs, like a thread of error, through the elaborate argument of Civiale, in his Chapter on the Dissolution of the Stone. See chap. iv. of his work, Du Traitement Médical de la Pierre.

(*c.*) Alkaline substances, it is urged, impair digestion. This objection was valid against the ruder methods of alkalising the urine formerly employed. But the acetates and citrates have no such effect. The introduction of these salts (and the bicarbonate) in recent times for the treatment of articular rheumatism, has afforded an immense field for watching their effects. Indeed the solvent treatment here recommended is identical with the prevailing mode of treating rheumatism, except that the dose is administered in a somewhat more dilute form. In the last eight years I have employed the bicarbonate, the acetate, and the citrate of potash, both in private and public practice, in doses of four, six, and eight drachms in the twenty-four hours, in a very large number of cases. The majority were cases of articular rheumatism; the remainder embraced a variety of slighter and more severe disorders—skin diseases, emphysema, diabetes, acute Bright's disease, etc. The urine was kept continuously alkaline for periods varying from a fortnight to three months, and in no instance were deleterious effects observed. In one patient with pulmonary emphysema, the urine was kept uninterruptedly alkaline for fourteen weeks, with marked improvement of the general health and steady increase of weight. In short, the acetate and citrate of potash have appeared to me about as harmless as so much sugar.

ON THE SOLVENT TREATMENT OF URIC-ACID CALCULI BY INJECTIONS INTO THE BLADDER.

It has been conceived that considerable advantages would be gained, in cases of vesical calculi, by injecting the solvent directly into the bladder, in a continuous stream, by means of a double-current catheter. The advantages chiefly counted on were :—the use of stronger solutions, and the employment of a greater mass of the solvent. In the case of uric-acid calculi, numerous experiments undertaken by myself show clearly that these advantages are illusory.

The mode of proceeding which I adopted, was to place a section of a uric-acid stone in a 10-ounce phial, and to pass over it at blood heat, a current of the solvent as large as the capacity of the urethra might be supposed to permit. The current was kept up for two or three hours continuously.

From experiments already recorded at p. 237, the maximum solvent power of the carbonated alkalies is ascertained to lie in solutions containing about 50 grains to the pint. A solution of *carbonate of potash* of this strength was passed over a fragment of uric acid weighing 57 grains, at the rate of forty-two pints per hour, for a period of three hours. The result was a dissolution at the rate of two grains per hour. This result, insignificant as it is, could probably not be approached in the living bladder on account of the mechanical difficulties to be overcome.

A solution of *carbonate of lithia*, containing 10 grains to the pint, with an hourly flow of 30 pints, dissolved less than one grain per hour. A solution of the same salt containing 20 grains to the pint, with an hourly flow of 26 pints, dissolved one and a quarter grain per hour.

Solutions of the following substances were also tried in a similar manner—namely, *borax*, *borax with liquor sodæ*, *double borate of potash and soda*, *common phosphate of soda*, *basic phosphate of soda*, and *potash soap:* but their solvent effects did not reach beyond a loss of weight of one or two grains in the hour.

Lime-water in a continuous current, at the rate of 30 pints per hour, dissolved a fragment weighing 86 grains, at the speed of one and a half grain per hour.

Seeing the very small results thus obtained, I proceeded to try the *caustic alkalies*, which are the most powerful known solvents of uric acid. But solutions, such as could be borne by the living bladder, of liquor potassæ and liquor sodæ (60 and 120 minims to the pint), did not dissolve more than about two grains per hour.

The general conclusion from these experiments* is, that under the most favourable conditions, and with the most effective solvents capable of being borne by the living bladder, no greater dissolution than one or two grains per hour can be accomplished in the case of uric-acid calculi. In actual practice the conditions would necessarily be much less favourable than in an experiment performed in the laboratory. A little consideration is sufficient to show that these results hold out no prospect of any useful practical application.

SOLVENT TREATMENT OF CYSTINE CALCULI.—Cystine is soluble both in the carbonates of the fixed alkalies and in the mineral acids. It may therefore be attacked, when existing as a calculus in the bladder, either by alkalising the urine as in the solvent treatment of uric acid, or by injecting acid solutions into the bladder.

Two experiments were performed with a view of testing the solubility of a cystine calculus in a solution of carbonate of potash containing 40 grains to the pint. The mean result, with a daily flow of three and six pints, showed a rate of dissolution equal to 20 per cent. of the weight of the stone in twenty-four hours. Cystine may therefore be regarded as being even more favourable to the application of the alkaline solvent treatment than uric acid.

SOLVENT TREATMENT OF OXALATE OF LIME CALCULI.—In the case reported at p. 246, alkalised urine flowed over the surface of a mulberry calculus for three months without producing the slightest show of solution. I also found that a solution of carbonate of potash containing 40 grains to the pint, passed over a mulberry calculus at the rate of six and eight pints in the twenty-four hours, had not the slightest solvent effect.

Better results, it was conceived, might be obtained by a solution of dilute nitric acid (which is the best solvent of oxalate of lime), employed so as to imitate injections into the bladder. A solution containing 120 minims of the concentrated acid to the pint, was passed over a mulberry calculus weighing 53 grains, at the rate of 24 pints per hour; and yet only half a grain was dissolved in an hour. We may conclude, from these experiments, that oxalate of lime calculi are unassailable by solvents applied in any known method.

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* The experiments here referred to are more fully described in the author's paper in the Medico-Chirurgical Transactions for 1865.

SOLVENT TREATMENT OF PHOSPHATIC CALCULI.—Phosphatic calculi were found quite unimpressible, as might have been expected, to solutions of carbonate of potash. Far more promising results were obtained by dilute nitric acid, used so as to imitate injections. A solution containing 60 minims of the commercial acid to the pint, was passed at blood-heat over a phosphatic stone weighing 153 grains, at the rate of 36 pints per hour. The loss of weight which followed amounted to 21 grains per hour. A modification of this proceeding was successfully employed, as is well known, by Sir B. Brodie in actual practice. My colleague, Mr. Southam, has recently tried the same method, and with the best results. The stone had been repeatedly crushed with the lithotrite; but fresh phosphatic concretions formed in the bladder as fast as the old ones were broken up; and it was found impossible to completely clear the bladder. In this difficulty an injection, containing two drachms of dilute nitric acid to a pint of water, was practised every day, or every second day. In the course of a short time the old fragments were completely dissolved, and the formation of new ones prevented. This method is evidently capable of wider application than is now made of it by surgeons.

CHAPTER IV.

CHYLOUS URINE.

PROUT—Stomach and Renal Diseases, 5th ed. p. 112.
RAYER—Maladies des Reins, tom. iii. p. 387.
BIRD—Urinary Deposits, 5th ed. p. 416.
BENCE JONES—Phil. Trans. 1850; and Med.-Chir. Trans., vols. xxxiii. and xxxvi.
BEALE—Urine and Urinary Deposits, p. 269.
WATERS—Med.-Chir. Trans., vol. xlv. p. 209.
CARTER— Ditto, ditto, p. 189.
PRIESTLEY—Edin. Med. Journ. 1856, p. 945.
BOUCHARDAT—Annuaire de Thérapeutique, 1862, p. 200.
PEARSE—Med.-Chir. Trans., vol. xxxiv. p. 127.
ACKERMANN—Deutsche Klinik, 1863, Nos. 23 and 24.
ISAACS—American Journ. of Med. Sci., April, 1860.
ELLIOTSON—Med. Times and Gaz., Sept. 19, 1857.
DUTT—Lancet, 1862, vol. ii. p. 87.
BEGBIE—Ed. Med. Journ., Aug. 1862.

THE disorder named by Prout chylous urine, is, properly speaking, a disease of tropical climates. It prevails endemically in the Mauritius, Isle of Bourbon, West Indies, the Brazils, and India. The cases met with among Europeans are almost confined to sailors, merchants, colonists, and others who have passed a portion of their lives in one of the above-named countries. The following account of the complaint is drawn up from an analysis of twenty-six cases, the sources of most of which are indicated at the head of the chapter.

In this disorder the urine is usually white and opaque, like milk; sometimes it has a faint rose tint, from a slight admixture of blood; and sometimes it is mixed with blood in clots.

On standing awhile, it sets spontaneously into a trembling coagulum, which after a time re-dissolves, and breaks up into flaky clots. Not unfrequently this coagulation takes place within the bladder, and occasions serious pain and difficulty in micturition. The milky appearance of chylous urine is due to the presence of a finely-divided fatty or oily matter. This is thrown up as a creamy layer after the urine has stood some hours. When chylous urine is agitated with ether, the fat is dissolved, and the secretion assumes the transparency and colour of healthy urine. The ethereal extract yields, on evaporation, a quantity of yellowish solid or oily uncrystallizable fat, resembling that which is found in the blood. Chylous urine is invariably coagulated by heat and nitric acid.* These united reactions indicate the presence of fibrine, fat, and albumen. Caseine, though specially looked for by Rayer, Guibourt, Pearse, and others, has never been authentically found in chylous urine; nor has sugar ever been found therein. The ordinary ingredients of healthy urine are present in their usual proportion, unless there be some superadded disease. The specific gravity is generally below the average. When examined microscopically, chylous urine is found to contain a variable number of granular nucleated corpuscles, like those of mucus or chyle; and generally, but not always, red blood disks. The fatty matter almost invariably occurs in the form of excessively minute granules (resembling the molecular base of the chyle), which are not resolvable into visible globules under the highest powers of the microscope. Occasionally, however, visible fat globules are found, as in the case recorded by Dr. Waters. Casts of the uriniferous tubes have never been found, though specially searched for by Bence Jones, Waters, Isaacs, Begbie, and others.

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* Bramwell's case (Edin. Med. Journ. 1858, p. 714) is excluded from consideration. It differs so greatly from all the other recorded cases, that it must be regarded as belonging to a different series, or as a case of imposition. In this case the urine did *not* coagulate spontaneously: it did *not* precipitate with heat and nitric acid: the fatty matter separated almost completely on simple standing: it contained *visible* fat globules $\frac{1}{4000}$ of an inch in diameter, and the fat extracted by ether crystallised like margaric acid; the patient had never been out of Scotland. In the want of coagulation with heat and nitric acid this case stands quite alone; in the other circumstances, if not altogether alone, it stands in a highly exceptional position. Dr. Barry's case (Beale's Archives, 1861, p. 46) is also rejected on account of the doubtful way in which it is reported. In the second ed. of Dr. Beale's work on urine and urinary deposits (1864), Barry's case is not alluded to.

Sometimes the urine is not chylous, but *lymphous*: that is, it contains albumen, and coagulates spontaneously, but the fat is absent, together with the opaque milky appearance which depends thereon. The coagulum in lymphous urine resembles calf's-foot or currant jelly.*

In this curious disorder, the urine resembles in every particular a mixture of ordinary urine with variable quantities of chyle or lymph: and a strong probability exists, as will be presently seen, that chylous and lymphous urines are, in fact, such mixtures.

The unnatural ingredients—albumen, fat, and fibrine—vary considerably in their relative proportions. The following table presents an abstract of nine analyses of chylous urine by different authors :—

	Quevenne. (Rayer, iii. 427.)	Rogers. Mean of 3 analyses. (Bird, p. 420.)	Bouchardat. (Annuaire, 1862, p. 201.)	Beale. (Archives, v. i. p. 12.)	Bence Jones. (Phil. Trans. 1850.) Mean of 2 analyses.	Dr. B. Edwards. (Med. Chir. Tr. v. xlv. p. 217.)	A. Gamgee. (Ed. Med. J., Aug. 1862.)
Fatty matter . .	1·90	1·10	1·30	1·39	0·79	0·99	0·20
Albumen . . .	0·70	0·33	0·20	1·30	1·40	0·60	0·17
Normal solids of the urine .	2·30	4·71	3·73	2·57	2·88	1·68	3·04
Water . . .	95·10	93·36	94·77	94·74	94·93	96·73	96·59
	100·00	100·00	100·00	100·00	100·00	100·00	100·00

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* Mr. Stocks, of Salford, sent to me, Nov. 18, 1864, a man named Williams, aged twenty-seven, who had never resided out of England. In 1862 this man was the subject of lymphous urine for about a month. He was at that time suffering from an extensively distributed, and severe, cutaneous disease of an eczematous character. Mr. Stocks gives the following description of the urinary symptoms. "There was great pain over the kidneys, in the perineum, and about the anus—defecation aggravating the latter much. No tenderness existed in the prostate. There was stillicidium urinæ, and frequent, painful, straining, micturition—half an ounce of urine passing at once. Masses resembling pieces of tripe, about the thickness of a lead pencil, were pulled out of the urethra two or three times a day for about a week. The urine itself was clear, highly albuminous, and, when allowed to stand, coagulated spontaneously into yellow transparent masses, floating in the fluid part of the urine, exactly like half-melted calf's-foot jelly. These masses again

The *course* of the disorder is marked by an irregularity and capriciousness which baffles explanation. The invasion is sometimes gradual; but more commonly it breaks out suddenly without previous warning or known cause. In other cases it comes on apparently after a fall, or in consequence of hard mental or bodily work. Its further progress is essentially intermittent; but it rarely happens that the intermissions follow any regular rule. An attack may last a few days, a few months, or many years. The intervals between the attacks vary similarly; the disorder may go on intermittingly for two, three, or more years, then cease for ten or more years, and be again renewed. The suspensions and renewals are generally quite abrupt, sometimes more gradual. During the remissions the urine returns to a perfectly normal state. Sometimes the attacks observe a certain periodicity. In one case it is related that the urine always became chylous for eight days previous to menstruation; in another, the recurrence almost always preceded or accompanied attacks of epilepsy or erysipelas. In Mr. Pearse's case the urine became chylous when the patient was suckling her children, and ceased to be so shortly after weaning them. It has been observed in several instances that an intercurrent disorder, such as a fit of the gout, hepatitis, carbuncle, inflammation of the lungs, severe ptyalism, has temporarily suspended the chylous condition of the urine. In other instances it dates its origin or renewal from some such attack.

There are also diurnal irregularities in regard to meals, exercise, and rest, which are inexplicably contradictory. As a rule, rest and fasting diminish or suspend the milkiness of the urine. In some cases the urine is chylous throughout the twenty-four hours, as in Dr. Waters's case: in others it is natural or lymphous on rising in the morning, and chylous during the remainder of the day, especially after dinner: in

became fluid in about twenty-four hours, leaving cobwebby fibres floating in the urine." When this man was seen by me he had lost the cutaneous eruption, and was able to follow his employment of warehouseman. Micturition was still unduly frequent; but the urine was free from fibrine and albumen. The only unnatural objects found after a careful microscopical examination were a few blood and pus corpuscles. Was this case an example of eczema invading the mucous membrane of the bladder?

Mr. Cubitt's case (cited by Beale) the urine was never chylous during the day, but only on rising in the morning. In Ackermann's case the urine became perfectly natural when the patient lay on his right side, and immediately resumed its chylous character when he stood up. Dr. Bence Jones found, in a case observed by him, that meals and exercise had a marked influence on the state of the urine. Shortly after a meal the urine became chylous: if the patient fasted and took exercise, the urine was lymphous: if he fasted and remained perfectly tranquil, it became natural. In Mr. Dutt's case the urine voided during the day was clear and free from chyle, while that voided during the night and in the morning was deeply loaded with it.

Illustrative Cases.—The following abstracts of cases will convey an idea of the character of the urine and the capricious course of the disorder:—

Case I.—A married woman, aged thirty, had been passing chylous urine about a year. Three specimens of her urine were submitted to Dr. Prout for examination, namely, one voided in the morning; another a little after breakfast; and a third in the evening.

The first specimen, voided in the morning, consisted of a solid jelly-like mass or coagulum of a pale amber colour. This coagulum was of an extremely delicate texture, and, on being submitted to a gentle pressure, or even allowed to drain, parted with a large proportion of a serous fluid of the colour above mentioned, and at the same time became exceedingly reduced in bulk, and assumed the appearance of a red fleshy-looking mass of a fibrous texture, which on examination was found to have all the properties of the fibrine of the blood, mixed with a few red particles of the same fluid. The specific gravity of the serous portion was 1019. Its smell was very faintly urinous; reaction neutral; it contained a large quantity of albumen.

The second specimen, voided after breakfast, resembled the first in its general character, but differed from it in some minor particulars. Thus, the serum was more of a whey colour; the fibrous coagulum was less, but more compact and firm, and held entangled in its texture a large proportion of the red particles of the blood. The specific gravity of the serum was only 1012, and it contained a considerable proportion of albumen.

The third specimen, voided in the evening, after an early dinner taken about noon, so closely resembled chyle in all respects, that Dr. P. was doubtful, if it had been brought to him as a specimen of that fluid, whether he should have discovered the imposition. It consisted of a solid coagulum of a white colour, and assuming the shape of the vessel, like blanc-mange. On being submitted to a gentle pressure and permitted to drain, the residual solid portion was, like that of the others, small in quantity, but whiter than the coagula of the other specimens. It was, however, intermixed with strings of a firmer consistence and of a red colour. The serous portion was white and opaque like milk, and on being heated and allowed to stand at rest for some time, threw up a substance on its surface resembling cream, and which, like cream, contained a considerable proportion of oily principle. Its specific gravity was 1017, and its smell was not urinous until it had been concentrated by evaporation; it was not coagulable by heat, though it contained abundance of albumen.

Dr. Prout had an opportunity of examining this woman's urine after fasting twenty-four hours. The coagulum was now much smaller in bulk, and seemed to contain more red particles. The serous portion was nearly transparent, and possessed in a considerable degree the colour and other sensible properties of urine. It contained albumen and abundance of urea.

This woman died emaciated, after suffering from the disease twenty years. (Prout on Stomach and Renal Diseases, 5th ed., p. 117.)

Case II.—The patient was a sailor, a native of Bermuda, treated by Dr. Waters in the Liverpool Northern Hospital. The characters of the urine are thus described. When first passed, it is white, with rather a pink tinge. It resembles new milk in appearance and somewhat in smell. It is perfectly free from urinous odour. After it has been passed for a short time it coagulates into a tremulous mass exactly resembling blancmange. The coagulum sooner or later disappears, entirely or in part, leaving the urine altogether fluid or partly clotted. After the urine has been standing some hours, a distinct deposit of florid blood is found at the bottom of the vessel, and the mass of fluid above assumes a perfectly white colour, showing that the pink appearance of the urine when first passed was due to the admixture of blood. There is in addition to the blood-deposit, a deposit of a somewhat slimy chararacter, having all the appearance of a mixture of pus and mucus. After standing some hours, a distinct thin layer of white fluid, exactly resembling cream, generally forms on the surface of the urine, the layer being thicker in some specimens than in others. The urine remains free from odour for some time, but at the end of three or four days it has a slightly urinous smell.

When first passed the urine is slightly acid or neutral, and soon becomes alkaline. Heat causes a precipitate of very fine particles. Nitric acid also produces a slight precipitate, but heat and nitric acid together cause a copious deposit. When boiled with liquor potassæ and sulphate of copper, there is no reduction of the copper to the state of suboxide. If the urine be agitated in a test-tube with an equal part of sulphuric ether and left to stand, a thin layer of fatty (?) matter is deposited on the surface of the urine and below the ether. The urine then becomes quite clear, and if removed by means of a syphon and boiled with nitric acid a copious deposit takes place.

When examined under the microscope the urine is found to contain blood, pus, and mucus corpuscles, with a large number of small fat globules. Many of these last are very minute, whilst others are larger. No casts of the uriniferous tubes, nor any other abnormal matters than those already mentioned were found in the deposit. The thin layer of cream-like fluid before alluded to consists entirely of oil globules. (Dr. Waters, Med. Chir. Trans., vol. xiv., p. 211.)

Contrasting with this description in the occasional absence of spontaneous coagulation and of visible fat globules, is the following account by Dr. Beale:—

CASE III.—The specimen of urine was passed in the morning. It was perfectly fluid, without any tendency to spontaneous coagulation, and had all the appearance of fresh milk. It had neither a urinous smell nor taste. Upon the addition of an equal volume of ether, it became perfectly clear.

Under the microscope the slight deposit which formed after standing some time, was found to consist of a small quantity of vesical epithelium, and some small slightly granular circular cells resembling chyle or lymph corpuscles. No oil globules could be detected on the surface of the urine or amongst the deposit, and the fatty matter, which was equally diffused throughout, was in a molecular or granular form. By examining the urine with the highest powers, only very minute granules could be detected. These exhibited molecular movements. In this case the urine was not, however, always uncoagulable, occasionally it coagulated even within the bladder. This case is exceptional in the circumstance that the urine was never chylous, except on rising in the morning. During the rest of the day it was always perfectly natural. The patient was a native of Norfolk, and does not appear ever to have been out of England. (Beale, Urine and Urinary Deposits, 2nd ed., p. 272.)

CASE IV.—A clergyman, aged forty, born in Bermuda, consulted Dr. Bence Jones in 1852. Ten years before, the urine became milky, and continued so for eight weeks; it then returned to its natural state without treatment. Five years after, the complaint returned. The patient passed clots and semi-solid masses with some difficulty. This second attack lasted two or three months, and then the urine became perfectly natural, and continued so for the succeeding four years. At the end of this period the disease returned, and had continued ever since, with the exception of an interval of three weeks. When the patient came under the care of Dr. B. Jones in 1852, the urine was milky, but it cleared with ether; it contained much albumen and some blood corpuscles, but no casts of tubes; its reaction was acid, specific gravity 1025. The patient stated that bodily or mental exertion (such as preaching on Sunday) produced the most intense milkiness of the urine. Usually the urine was milky on going to bed; it was clear in the morning, until an hour after breakfast; the whiteness then increased according to the degree of exercise taken. He dined at one, and then, with rest, the urine became clear, and continued so until he took his afternoon walk, when the whiteness returned. He had tried all sorts of tonics, buchu and iron, and had taken gallic acid on this and the previous occasions without advantage. (Bence Jones, Med. Chir. Trans., vol. xxxvi., p. 91.)

CASE V.—A lady, aged sixty-four, born in India, where she had resided for some years, came under the care of Dr. Elliotson. The urine became for the first time milky *nine years* after her return to England. It continued milky, in spite of various remedies, for about a year, when it suddenly resumed its natural appearance on the third morning after she had commenced to take a daily bath in the sea. The urine then remained clear for thirteen years (eighteen months of which were passed in India). At the end of this period she had a severe inflammation of the lungs, for which she was bled, and took calomel. In a month after this attack the urine again became chylous, and continued so for two years. At this time she suffered from a severe mental shock, and for the space of one month the milkiness of the urine was suspended; it then returned with as much intensity as ever. From this date the urine continued milky without one day's intermission for eleven years. The milkiness was then, once more, suspended for six weeks in consequence of a carbuncle; then it went on again for two years, when she had a second attack of inflammation of the lungs, which laid her up for six months. During this illness the chylous state of the urine was again suspended; but has since returned, and still continues (1857). The patient is a very stout person, and very nervous. All remedies have been unavailing. The disease has continued, with intermissions, for eight-and-twenty years. (Elliotson, Med. Times and Gaz. 1857, ii. 287.)

CASE VI.—An English gentleman, aged forty, a teacher of languages, settled in Rostock (Germany), came under the notice of Dr. Ackermann. In his youth, this gentleman had travelled for two years in the Brazils and Buenos Ayres. While in South America he suffered from a slight hydrocele,* which disappeared on his return to Europe.

He had been settled in Rostock for eleven years, and was strong and very healthy. Midsummer, 1858, he took the measles, which left behind a slight bronchial catarrh. This catarrh lingered on till February, 1859; when one day he observed his urine to be milky. A few hours before this occurrence he felt out of sorts and shivered, but next morning he was well again. The urine, however, continued milky, and five weeks after, leeches were applied to the left loin on account of a tenderness which existed in that region. During their application the patient lay on his right side for two hours, and immediately on rising he made water, which, much to his own and his physician's astonishment, was perfectly normal, clear, and of a deep yellow colour. Nevertheless, at the next micturition, the urine was found milky again. A few days after, the patient repeated the experiment: he emptied the bladder and lay for an hour on his right side, and again the urine appeared clear, and contained only traces of albumen. Similar results were obtained many times after the same experiment. If perfect rest on the right side was broken even for a few minutes the urine was distinctly chylous at the end of the experiment. A counter-experiment, in which the patient lay on his left (instead of right) side, showed still more distinctly the effect of the posture on the right side. On the 6th of May the patient lay for an hour on his left side; but the urine which he made on rising was strongly chylous and contained blood. At a later period this influence of lying on one or the other side became less marked and constant. But throughout the complaint, rest in the horizontal position had invariably the effect of diminishing the chylous condition of the urine. The morning urine, after the rest of sleep, was always the least milky; and that of the evening, after the fatigues of the day, the most so. The general health was only slightly affected. He was a little less capable of exertion, more easily fatigued, very sensitive to cold, and somewhat depressed in mind; there was also a dull pain in the left lumbar region. The exhaustion appeared to increase as the disease continued, but he was not compelled to suspend his somewhat arduous occupation for more than a day or two. He noted that a hœmorrhoidal flux with which he had been previously frequently affected, ceased entirely from the moment the urine became milky. In July and August the patient spent a month at the sea-baths of Warnemünde, where he led a very quiet life, but did not bathe. Here the urine suddenly became clear and normal, and continued so for a fortnight; but before he left, it became, as suddenly, intensely milky again. At a still later period more frequent variations in the chylous and non-chylous condition of the urine were observed than in the beginning; but no intermission as long as that noted at Warnemünde occurred again. It often happened that amid a long series of chylous emissions a normal one would be suddenly interposed, and it was not always possible to find any cause for this sudden change in the circumstances of the patient.

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* Query—Was not this supposed hydrocele an affection of the scrotal lymphatics, as in Carter's cases, to be presently described?

It was observed that the skin was markedly less disposed to sweating than previously, and sometimes there was a disagreeable dryness of the cutaneous surface. The urine was notably increased in quantity, especially in the earlier periods, when it exceeded five pints in the twenty-four hours.

The examination of the urine yielded the following results:—It was opaque, almost milk-white with a tinge of red, reaction acid, with a stale sweetish odour. It coagulated spontaneously, sometimes after standing a few minutes, sometimes after several hours. It never gelatinised within the urinary passages. It also coagulated with heat and with nitric acid. When it was allowed to rest for eighteen hours in a glass, a thin, perfectly white layer gathered on the top, and a reddish deposit sank to the bottom. In the former a vast quantity of fat-molecules were found; in the latter, blood corpuscles and small dark red clumps of blood. Ether cleared the urine almost completely, and the extracted fat was solid at ordinary temperatures. When the urine was boiled, it passed through a filter perfectly clear, and possessing all the qualities of healthy urine—the fatty matter having been entirely retained on the filter by the coagulated albumen.

The table on the following page exhibits the proportion of fat and albumen at different times of the day.

Various remedies—among them gallic acid—were tried in this case with little or no evidence of success. In the beginning of December, 1859, he left off all medicine, finding them of no effect on his urine, and feeling his general health satisfactory. Toward the end of January, 1860, he found himself one evening, after a very heavy day, greatly exhausted, and chilly, and therefore went earlier than usual to bed. The urine on this evening was strongly chylous. Next morning, on the contrary, the urine was perfectly normal; the succeeding emissions were similarly healthy, and from that day forth the disorder did not return. For three years he has continued to pass perfectly natural urine. A few days after this

TABLE EXHIBITING THE PROPORTION OF FAT AND ALBUMEN AT DIFFERENT TIMES OF THE DAY IN ACKERMANN'S CASE OF CHYLOUS URINE.

Date.	Hour of micturition.	Antecedent condition.	Specific gravity.	Colour.	Fat per cent.	Albumen per cent.	Sediment.
Oct. 15	7 a.m.	Sleep.	1024	... ...	0	0	None.
,, 16	11 a.m.	2 hours sitting.	1025	yellowish white	0·32	0·506	Fibrinous flakes.
,, 18	11 a.m.	2 hours tuition out of the house.	1011	white, with little yellow	0·34	0·145	A few pink fibrine flakes.
,, 19	noon.	3 hours of the same.	1024	... ...	0·47	0·426	Shreds of fibrine.
,, 20	5 p.m.	3 hours of the same.	1018	light yellowish white	0·26	0·968	Brownish-red clumps.
,, 21	6 p.m.	3 hours of the same.	1010	Do.	0·09	0·420	None.
,, 23	10 p.m.	3 hours sitting and half-hour reclining.	1011	... ...	0·24	0·202	None.
,, 24	7 a.m.	Sleep.	1021	... ...	0·10	0·647	Shreds of fibrine.

favourable termination, the hæmorrhoidal flux returned, and has continued since with its former frequency.* (Ackermmann, Deutsche Klinik, 1863. Nos. 23 and 24.)

The general health of persons affected with chylous urine, suffers in varying degrees. Some persons preserve their embonpoint undiminished; but the larger number are markedly emaciated. The patients generally complain of lassitude, incapacity for exertion, pains in the loins and the epigastrium. The unnatural drain of the nutritive material explains most of these symptoms. Sometimes there is an excessive appetite: more commonly the appetite is natural or indifferent. The long series of years, during which persons may void chylous urine, without serious impairment of their health, shows the comparative innocuousness of the complaint. In Quevenne's case (cited by Rayer) the patient, a native of the Isle of Bourbon, commenced to pass chylous urine at the age of 25. From this period to the age of 73 she constantly passed chylous urine. At 73 the urine became natural, and the patient thought herself cured; but after about 14 months, the urine became again as chylous as ever, and continued so until she reached the age of 78, beyond which the report does not go. In Dr. Elliotson's case the disease lasted, off and on, for 28 years, without seriously affecting the health. When death has occurred in cases of chylous urine, it has been occasioned by some independent malady. In Dr. Priestley's case the kidneys presented the fatty form of Bright's disease, and the lungs were tuberculous. At his Gulstonian lectures, in 1831, Dr. Prout exhibited the kidneys of a girl of 15 who had been passing chylous urine. She was said to have died of inflammation of the bowels: the kidneys were perfectly healthy. Dr. Isaacs had an opportunity of examining the body of a sailor, who during life had been in the habit of passing chylous urine, and who had died of general tuberculosis. The kidneys contained a few nodules of secondary tubercle, but were otherwise perfectly healthy.

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* This case came under the notice of Dr. Thudichum in 1864. The patient had returned to England, and the chylous state of the urine had re-appeared, in consequence, as the patient believed, of higher living. (Brit. Med. Journ. *1864, p. 611.*)

Etiology.—Chylous urine prevails mostly in youth and middle age. Of 26 cases collected by me, 3 were under twenty; 7 between twenty and thirty; 7 between thirty and forty; 2 between forty and fifty; and 3 over fifty. The youngest example is mentioned by Prout, in a male infant of 18 months; the oldest in Quevenne's case, in which the patient reached the age of 78 years.

Of these 26 cases, 17 were males and 9 females: but it appears, that in the countries where the disorder is endemic, it is more common among women than men. The greater frequency of it among men in the European cases is explained by the far greater number of men, who, as sailors, merchants, colonists, etc., pass a portion of their lives in tropical climates.

As to the causes of the disease, the only one made out satisfactorily is residence in certain tropical countries. Twenty-one cases (out of 26) were persons who had been born, or had passed a portion of their lives, in the Mauritius, Isle of Bourbon, Brazil, West Indies, or India.*

The best authenticated cases in persons who have never been out of Europe are—a case related by Prout; Mr. Gossett's case, cited by Bird; and Mr. Cubitt's case, cited by Beale.

The state of the blood has been examined by Guibourt and Bence Jones. Neither of these observers found a milky state of the blood-serum. Guibourt (cited by Rayer) obtained nearly twice as much fat from the blood of a Brazilian affected with chylous urine as from healthy blood. Bence Jones, on the contrary, found in the blood of a person whose urine was milky both before and after the bleeding, no increased proportion of fat.

Pathology.—It has been generally assumed that the fat albumen and fibrine of chylous urine are derived from the blood, and pass into the urine through the kidneys. Dr. Prout says:—"The proximate cause of this affection seems to lie partly in the assimilating organs, and partly in the kidneys. The chyle, from some derangement in the processes of assimilation, is not raised to the blood standard, and consequently, being unfit for the future purposes of the economy is, agreeably to a law of the economy, ejected through the kidneys; but these organs instead of disorganising it, or reducing it to the crystallised state, as usual, permit it to pass through them unchanged."

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* The endemic prevalence of chylous urine in these countries is thoroughly attested. The various speakers in the discussion at the Medical Society of Rio Janeiro, reported by Rayer, allude to the disorder as a common one among negroes. Dr. Prout states that Mr. Thomas, a practitioner from Barbadoes, informed him that he had seen at least a dozen well-marked cases in negroes in a practice of ten years. Dr. Carter also says that the more ordinary forms of chylous urine are "not uncommon" in Bombay.

But any view which supposes that the unnatural ingredients of chylous urine are derived from the kidneys presents great difficulties. The rapid alternation of urine intensely chylous or lymphous, with perfectly natural urine (sometimes witnessed within intervals of an hour or two) seems incompatible with such a supposition. It is also incredible that blood, albumen and fibrine should pass from the blood into the urine through the kidneys without being accompanied with casts of the uriniferous tubes. The absence of organic disease in the kidneys, and of any clearly made-out derangement in the composition of the blood, also militates against such a view.

A new and unexpected light has been thrown on the nature of this singular malady, by some cases published by Prof. Carter, of Bombay, which appear to indicate, that a direct communication does, in reality, exist between some part of the lacteal or lymphatic system and the urinary passages. These cases and the remarks of Dr. Carter thereupon are well-deserving of attention.

Dr. Carter's first case.—This was a Parsee youth in whom the inguinal glands were greatly enlarged, soft and doughy to the touch, but not painful. On the cutaneous surface of the thigh, a few inches below Poupart's ligament, was a small, hardly perceptible pimple, from which there occasionally issued a milky fluid, and sometimes so copiously, that in the course of the day a pint has been collected. Pressure just above the spot caused the flow to cease. The discharge commenced six months before: it lasted two or three days and then ceased, pressure having been applied; it reappeared after an interval of a month, and again stopped after a few days. The discharge reappeared a third time, when the patient came under the observation of Dr. Carter. Before the discharge came on, the glands in the groin became tumid and rather painful. The fluid collected from this man's thigh presented in perfection the characters of chyle. It coagulated in about five minutes; it separated a few hours afterwards into clot and serum, the latter being milky in appearance. At a later period the whole became again fluid. Under the microscope blood corpuscles and chyle corpuscles were found. In this man the *urine* never became chylous.

Dr. Carter's second case.—This was an adult Hindoo, who became a hospital out-patient, under Dr. Carter's care, on August 23rd, 1859, on account of an affection of the scrotum. The skin of this part was corrugated in a peculiar way, thickened and studded with numerous small tubercles, which were soft to the touch, and when punctured, freely discharged a chylous fluid. The inguinal glands on both sides were much enlarged, soft and doughy to the touch, and they diminished in size under pressure.

The scrotum began to enlarge four months before, and after a time the peculiar corrugation of the skin appeared. The milky discharge occurred occasionally and spontaneously, and intermitted. It did not issue from one spot but from several. When it ceased, and sometimes also during its continuance, the urine became chylous. The tumefaction of the inguinal glands seemed to alternate with the appearance of chyle in the urine. The parts also became tumefied two or three hours after a full meal, and then subsided.

The urine, when chylous, coagulated spontaneously, and the coagulum reddened sensibly on exposure to the air. This reddening was however more decided in the coagulum from the scrotal chyle, which changed in a few minutes to a blood red. Under the microscope the urine was found to contain, besides blood corpuscles, nucleated granular cells exactly resembling chyle corpuscles. Various plans of treatment were adopted, including very large doses of gallic acid, without effect.

Dr. Carter's third case was one of ordinary chylous urine, without any visible affection of the external lymphatics. The general characters and the microscopic examination of the urine appeared to establish the identity of the foreign ingredients found therein with those of the chyle-like fluid discharged from the external surface on the preceding cases.

Dr. Carter applies these facts to the phenomena of chylous urine in the following manner :—"We may suppose that distension of the delicate lymphatics and lacteals in the lumbar region is at length followed by exudation of their contents at one or more points ; or rupture taking place, a fistulous orifice remains, which gives free exit to the chyle or lymph at times of recurring distension ; or an abnormal reservoir (receptaculum) may be formed, which periodically discharges its contents into the pelvis of the kidney, ureter, or bladder. The cases before related evince that such a condition of the lymphatic vessels, accompanied with enlargement and increased functions of the corresponding glands, does occur ; that the flow of chyle may be reversed, or regurgitation may occur; and with this state, the urine may be chylous."*

Additional evidence in favour of this hypothesis may be deduced from the increased quantity of urine which has been observed in several of these cases, as in Ackermann's. In Dr. Waters's case the mean daily quantity of urine during the first ten days, when the disorder was at its height, was 67 ounces; in the last ten days, when it was subsiding, the mean daily quantity was only 47 ounces.

The prevalence of the disorder in certain countries, and the close connection which Rayer showed to exist between it and the endemic hæmaturia of the same countries, leads conjecture a step farther. It has been recently demonstrated by Griesinger that the endemic hæmaturia of Egypt owes its origin to the ravages of a minute parasite—the Bilharzia hæmatobia—which lodges its ova in the mucous membrane of the urinary passages. A similar demonstration has been made by Dr. Harley with respect to the endemic hæmaturia of the Cape of Good Hope (see BILHARZIA); and we may infer a similar origin to the endemic hæmaturia of the Mauritius, Brazil, and other countries. On these grounds, it may be conjectured that chylous urine owes its origin, in like manner, to the destructive operations of some parasite whose seat of election is the lymphatic system of the urinary passages, and that in the course of the development of its ova a communication is opened between the lymphatic channels and the urinary passages. At any rate, it is worth investigating whether parasitic ova may not be discovered occasionally in the deposit of chylous urine.†

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* Four years previously to the publication of Dr. Carter's paper, a similar view seems to have been propounded by Gubler in a paper entitled *Hæmaturie de l'Isle de France, envisagée comme une lymphorrhagie de l'appareil uropoëtique* (Memoirs of the Soc. de Biologie for 1858). I have not been able to consult the original paper, but an abstract is supplied in Canstatt's Jahresb. for 1858, of which the following is the closing sentence. "Gubler indicates chylous urine (of the Isle of France) as a diabetes lymphaticus, or a lymphorrhagia renalis." He rests his theory on the identity of the micro-chemical characters of chylous urine with that of chyle, and on the prevalence of dilatations of the external lymphatics in the countries where chylous urine is endemic.

† Dr. A. B. Buchanan, of Glasgow, has recently given an account of a woman, æt. forty-six, a native of Glasgow, in whom a large quantity of milky fluid, exactly resembling chyle, flowed interruptedly for years, from a

Treatment.—Hitherto the treatment of this disorder has proved very unsatisfactory. It generally persists in spite of every remedy, or disappears without any. The physicians of Rio chiefly recommend salt-water baths, and iron internally. Mineral and vegetable astringents have been tried repeatedly with small evidence of success. The best results have followed large doses of gallic acid. Dr. Waters and Dr. Bence Jones gave from one to two drachms a-day.

Dr. Bunyan, of George Town, British Guiana (Lancet, 1846, I. 95) relates a very interesting case, in which the disease had lasted ten months. Various remedies were tried without success. On the advice of an old negress, the patient took a decoction of mangrove bark (Rhizophora racemosa), in ounce doses, four times a day. In seven days, he was so greatly improved that he discontinued the medicine for two days, when the symptoms returned. The medicine was resumed in increased quantity, and continued for several days, until all the symptoms had entirely disappeared. Afterwards he suffered two returns of his disorder, which were immediately cut short by the use of the mangrove bark.

If further inquiry should show that chylous urine has a parasitic origin, anthelmintic remedies, especially turpentine, may be tried with a prospect of success. The effect of diet was investigated by Dr. Bence Jones. He found that the urine was somewhat less chylous with vegetable than with animal food; he also found that the pressure of a tight belt round the loins relieved the pains in the lumbar regions, and seemed to improve the condition of the urine a little.

thickened patch of skin on the inside of the thigh. This case resembled Carter's cases, except in the absence of an enlargement of the lymphatic glands. He also cites two other similar cases in Europeans. He proposes a new theory for the complaint and for chylous urine. Rejecting the theory of a leak in the lymphatic system as anatomically impossible and pathologically unnecessary, he contends that the fatty matter is secreted from the blood "by the morbid activity of multitudes of epithelial cells, the functions of which have become perverted." In chylous urine Dr. Buchanan believes that a similar disease to this exists in some part of the urinary mucous membrane. (Med. Chir. Trans. vol. xlvi., p. 57).

PART III.

ORGANIC DISEASES OF THE KIDNEYS.

CHAPTER I.

CONGESTION OF THE KIDNEYS.

ROBINSON—Med. Chir. Trans. 1843, p. 51.
FRERICHS—Die Bright'sche Nierenkrankheit, 1851.
HERMANN—Sitzungsberichte der mathem-naturw. Classe der Kais. Akad. Vienna, 1861, p. 26.
OVERBECK—Ueber den Eiweissharn, ibid., Feb. 1863.
BOUILLAUD—Archives Générales. 4me Série, Tom. xvii., p. 99.
JOHNSON—Diseases of the Kidney. Lond. 1852.
VIRCHOW—Archiv f. path. Anat. Band iv.
TRAUBE—Ueber den Zusammenhang von Herz-und Nieren-Krankheiten. Berlin, 1856.
BAMBERGER—Archiv f. path. Anat. Bd. xi., p. 16.
ROSENSTEIN, Path. u. Therap. d. Nieren-Krankheiten. Berlin, 1863.

UNDER the title of congestion of the kidneys, I propose to consider those less serious, and for the most part secondary, renal derangements which are occasioned either by an undue determination of blood to the organs (active congestion), or some mechanical obstruction to the return of blood from the organs (passive congestion).

Renal congestion, both active and passive, if sufficiently intense, is attended by the presence of albumen in the urine (generally in small quantity), sometimes with blood, and casts of the uriniferous tubes. Dropsy is not a symptom proper to renal congestion; when present it depends on other causes, commonly heart or lung disease.

Active congestion is produced by—over-doses of certain irritants (cantharides, turpentine); by exposure to cold; it is a common incident in all febrile and inflammatory complaints; it occurs in saccharine diabetes; probably also in some cases of hypertrophy of the left ventricle.

Passive congestion accompanies—regurgitant heart disease; obstructions in the lungs (emphysema, pleuritic effusion); pressure on the emulgent veins or inferior cava (pregnancy, abdominal tumours).

If the determining cause of the congestion be a persistent one—as in valvular heart-disease or diabetes, organic changes are at length produced in the kidneys, which bear a strong resemblance to, if they are not identical with, certain forms of Bright's disease.

Accordingly, several of the conditions here considered, have been arranged by other writers (Johnson, Frerichs, Griesinger, Bamberger) among the varieties of Bright's disease. But although there are unquestionable affinities between the two classes of cases, there are also differences so marked, in their symptoms, progress, and general clinical history, that it only tends to confusion to unite them under one heading.

It will greatly facilitate our comprehension of the relations subsisting between certain changes in the composition of the urine, and certain disturbances of the renal circulation, if we take a review of the experimental researches which have been made in this direction.

Mr. George Robinson was the first to demonstrate, that a complete or partial impediment to the return of blood by the renal veins caused albumen, blood, and sometimes fibrine to appear in the urine. He operated solely on rabbits. In one set of experiments, he placed a tight ligature round the renal vein: in a second set, the obstruction was made incomplete—a certain amount of blood being still permitted to circulate through the kidneys. In both these sets of experiments the urine invariably became more or less albuminous, and in most cases bloody. The kidney, of which the vein had been thus obstructed, was in every instance found heavier than its uninjured fellow. The proportion between them varied from 1½ : 1 to 3 : 1.

Frerichs repeated these experiments on dogs, rabbits, a cat and a frog, with identical results. In four out of ten experiments, he also detected casts of tubes in the urine, and, in one, renal epithelium.

These experiments admit of easy explanation. The blood accumulates behind the impediment, and causes an increased lateral pressure upon the walls of the renal vein and its branches. This tension is transmitted backward to the renal capillaries, which are thereby distended, and their walls attenuated, creating a condition highly favourable to the transudation of the serous constituents of the blood through their coats. If the tension be sufficiently great, actual rupture takes place, and blood corpuscles escape with the albumen into the urine. It is probable that these consequences take effect earliest, and in greatest power, in the Malpighian clusters, where there exist anatomical facilities for ready passage of blood into the urine.

Increased pressure in the *arterial* system does not so easily cause albumen and blood to appear in the urine. Robinson sought to test the effect of increased arterial pressure on the composition of the urine, by directing a stronger stream of blood than natural into the kidneys. First he removed one kidney, thinking that the physiological determination to the other might suffice to cause albuminuria. The experiment was repeated five times, and only in one instance did the urine become albuminous. He then removed one of the kidneys and tied the abdominal aorta below the origin of the renal arteries.* In this way the utmost impulsion of blood into the remaining kidney was obtained, and both blood and albumen invariably made their appearance in the urine. His seventh experiment is a fair sample of his results.

Expt. 7. The left kidney of a middle-sized rabbit was removed, and weighed 54 grains. The aorta was then tied below the origin of the renal arteries. The animal was killed at the end of two hours. The right kidney weighed 85 grains; it contained six or seven ecchymoses of various extent. The bladder contained about a drachm of urine, which was bloody and albuminous (l. c. p. 79).

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* Tying the abdominal aorta without removing one of the kidneys was performed twice by Robinson on weak animals; in one only did albumen appear in the urine. Frerichs states that he could only find traces of albumen in a few cases after such an operation. Meyer, on the other hand, saw abundant albuminuria follow this operation.

These results have been confirmed by Frerichs and Meyer.

In the experiments of Hermann and Overbeck, another method of inducing artificial albuminuria is pointed out. Hermann's method consisted in tying up the renal arteries for a short time, and then removing the ligature. The urine which was secreted after the re-establishment of the circulation was always found albuminous. Overbeck interrupted the circulation in other ways. In one set of experiments, he blew up a bladder previously introduced empty into the heart: in the second set, asphyxia (and consequent arrest of the blood-current) was produced by compressing the trachea. In the former case the obstruction was maintained for about a minute, and in the latter for four minutes. In both classes of experiments, the urine which first flowed after the renewal of the circulation was invariably albuminous, and often bloody. The albuminuria thus provoked, generally lasted a few hours, and then passed away. When desquamation of the renal epithelium occurred, it always *followed* the appearance of the albumen. It could not therefore be the cause of it, as Johnson surmised to be the case in the albuminuria of Bright's disease.

To explain the results obtained by Hermann and Overbeck, it must be supposed that the temporary stoppage of the blood-current created an obstacle in the renal capillaries—probably an accumulation of blood corpuscles in the Malpighian tufts—which, when the circulation was restored, operated to raise the pressure in the minute arteries; in other words, it produced active congestion of sufficient intensity to cause albumen and blood to appear in the urine.

My purpose in calling attention to these researches is to show, that simple hyperæmia or congestion of the kidneys (without inflammation), either from increased impulsion of blood into the kidneys, or from obstruction to the return of blood from the kidneys, is sufficient to determine the appearance of albumen and blood and even fibrinous casts in the urine.

An impeded circulation through the kidneys cannot however long persist, without inducing serious and permanent structural changes in the organs. The presence of blood corpuscles, and fibrinous plugs, in the delicate tubular structures, must at length occasion more or less extensive destruction of these structures; and the continued hyperæmia must derange the nutrition of the glandular elements. How far these changes are of an inflammatory nature, cannot be precisely indicated. One of the most important results of a long continuance of this state of things appears to be, an excessive production of adventitious connective tissue, which eventually passes on to contraction and atrophy. To call these changes, "nephritis," is to use a term, which, to say the least, is calculated to mislead.

ACTIVE CONGESTION.

(*Catarrhal Nephritis of Virchow.*)

In the course of eruptive and continued fevers, of croup, diphtheria, cholera, erysipelas, pyæmia, acute rheumatism, pneumonia, and other inflammatory diseases,* the kidneys partake in the general hyperæmia of the internal organs. Not unfrequently, however, they are the seat of a disproportionate determination of blood, and albumen appears in the urine. Generally speaking, the amount of albumen, in such cases, is a mere trace, but sometimes it is more abundant, and accompanied with a few blood corpuscles, transparent casts of tubes, and scattered renal epithelium. There may be, at the same time, some tenderness in the loins. As soon as defervescence commences, the albumen diminishes, and in a few days vanishes altogether.

The pathological state here described, differs from genuine Bright's disease (which may likewise arise in connection with the same febrile maladies) in the absence of anasarca, in the undiminished excretion of urea, and in the period of its invasion. Albuminuria from congestion coincides with the acme of the pyrexia, and subsides therewith. Genuine Bright's disease, on the contrary, shows itself as a sequela, toward the close of the pyrexial stage or the commencement of convalescence.

An examination of the kidneys of persons who have died from the primary fever while labouring under renal congestion, reveals an enlarged and engorged condition of the organs, with minute ecchymoses; and, according to Virchow, there exists a catarrhal state of the canals of the pyramids, of which the chief incidents are, in addition to hyperæmia, detachment and disintegration of the epithelium of the straight tubes. The detached epithelium sometimes shows signs of fatty changes.

The frequency of this complication in zymotic diseases, varies in different epidemics. Rosenstein states that in a severe typhus epidemic, witnessed by him in 1857, the majority of the patients had transient albuminuria, with casts of tubes, and yet no serious consequences followed therefrom. In the sporadic typhoid of this city, albuminuria is decidedly rare.

Active renal congestion of a catarrhal nature, may also arise independently of any specific fever, simply from exposure to cold. Such cases are not very common, or perhaps, as Rosenstein suggests, they are often overlooked. The symptoms resemble those of a simple febricula, and, unless the urine chance to be examined, the disorder will probably be passed over as such. The following example is from Rosenstein :—

A. B., æt. 39, previously healthy, experienced on the afternoon of the 7th of October a chill, followed by heat and severe pains in the renal region, which were accompanied with vomiting. When seen, she was in a high fever, pulse 120, very thirsty, and without appetite. The urine was scanty, acid, albuminous; after standing, it deposited a sediment composed of uric acid, blood corpuscles, epithelial casts, and free epithelium. Pressure on the renal region caused pain. She was cupped on the loins. On the following day the urine measured 27 ounces, specific gravity 1026, otherwise as before reported. On the 16th, the pulse was 92, skin moist and perspiring, general condition good. Urine in twenty-four hours, 28 ounces, specific gravity 1025, acid, free from albumen, containing only a few casts. On the succeeding days increased diuresis set in, with diminished specific gravity of the urine. The urine continued free from albumen and formed elements (p. 65).

A case of a similar nature, but running a more chronic course, was recently treated by myself, in conjunction with my friend Mr. Mellor.

The patient was a young lady of 26, who had been subject, for several years, to frequently recurring attacks of subacute articular rheumatism, which kept her in a continuously weak state of health. On April 14th she took cold through walking in the wet, and was seized with tonsillitis. As this subsided, the urine was noticed to be bloody and to contain albumen. On the 15th of May I saw her for the first time. She was very pale and thin; there was considerable fever, pulse 108, the loins were painful and very tender on pressure, skin dry, with a tendency to frequent vomiting. Micturition was very frequent (20 times a day); the urine amounted to three pints in the twenty-four hours, specific gravity 1010; it contained a good deal of blood and albumen, and deposited uric acid very abundantly. The copious deposit which subsided when the urine was left in repose, contained numerous large transparent casts (some studded with epithelium) and much free renal epithelium. Neither casts nor epithelium showed any signs of fatty degeneration. There were also found a large number of pyelitic cells. Not a particle of dropsy or anasarca existed in any part.

The patient was dry-cupped over the loins, after which hot poultices were directed to be kept (frequently renewed) to the same region; a compound jalap powder was administered, and a citrate of potash mixture.

In four days the fever subsided, the pains disappeared, and the skin became moist. At the same time, the urine was far less frequently passed, and it contained much less blood, albumen, and casts. It still continued abundant in quantity, and deposited uric acid very copiously.

In the course of four weeks, convalescence was so decidedly established, that the patient was allowed to sit up. The albumen now scarcely exceeded "a haze" with nitric acid. She was put upon a phosphoric acid mixture, combined with phosphate of iron.

On June 24th the patient suffered a relapse. She was again confined to bed, and the previous treatment put in force. In a few days the feverish symptoms passed off; but a good deal of blood, albumen, and renal derivatives continued to be discharged. She was now put upon gradually-increasing doses of dilute sulphuric acid, with most excellent effect. The urine steadily resumed its natural characters, and the patient's appetite and strength began to return.

On the 24th of July the urine had become free from albumen and blood, and convalescence was thoroughly established.

The case was, from the beginning, regarded as distinct from genuine Bright's disease, and considered as presenting the features of a catarrhal (rheumatic ?) condition of the pyramidal parts of the kidneys, combined with some degree of subacute pyelitis. The total absence of anasarca, and the general portraiture of the complaint, forbade the idea of acute Bright's disease; while the state of the urine and the progress of the case appeared inconsistent with the chronic forms of that formidable disorder.

Certain irritants—cantharides, turpentine, cubebs, copaiba, and nitrate of potash—act as special stimuli of the urinary organs; and excite, when administered in excessive doses, hæmorrhage from the kidneys and the lower urinary passages. Johnson relates an instance in which half an ounce of turpentine was taken for the expulsion of tape-worm. In a few hours the urine was bloody, and in the deposit "blood casts" were discovered, together with a few small inflammatory cells, but no epithelium. Six days after, the urine contained less blood and albumen. The casts of tubes were still visible, and contained, besides the blood corpuscles, a large proportion of inflammatory cells about twice the size of the blood corpuscles.* The patient continued to pass more or less blood for some days longer. On the sixteenth day the urine was free from albumen and blood (p. 488).

Bouillaud examined the effects of cantharides acting through the skin. He states that, almost constantly, when large blisters were applied to scarified portions of the skin, albumen appeared in the urine. After death, he found the mucous membrane of the pelvis and ureters, in other cases that of the bladder, injected, and covered here and there with false membranes. The kidneys were generally strongly congested and studded with minute ecchymoses. Albuminuria after cantharides usually disappeared in two or three days: in a few cases it lasted four weeks.

Two cases of poisoning by sulphuric acid are related by Leyden and Munk, in which albumen and casts appeared in the urine.†

Frerichs enumerates irritants of this class among the exciting causes of genuine Bright's disease; and brings forward two cases by Reinhardt, in which abuse of copaiba and cubebs were followed by renal degeneration, which, in one of them, proved fatal. These cases are, however, as Rosenstein points out, inconclusive, because it is probable that the kidneys were already diseased before the use of the irritants was commenced. There is no sufficient proof that genuine Bright's disease has really ever followed an overdose of any of these stimulants.

In a previous section, it has been mentioned that in the later periods of diabetes albumen not unfrequently appears in the urine. The excessive action of the kidneys in this disease, keeps up a constant congestion of the organs; and, in the course of time, permanent anatomical changes follow—degeneration of the epithelium, development of cysts, and other structural alterations, which are sometimes classified with genuine Bright's disease.

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* Were not these renal epithelia?

† Archiv. f. path. Anat. Bd. xxii. p. 237.

There is yet one other condition which seems capable, in rare instances, of producing an active congestion of the kidneys, sufficiently intense to determine albuminuria. In the compensatory hypertrophy of the left ventricle, which follows aortic regurgitant disease, the propulsion of blood into the aorta (when the orifice is patulous) takes place with very great force; and the tension of the arterial system at the close of the ventricular systole, rises considerably above the normal maximum, as is indicated by the full resistent pulse.* Practically, however, albuminuria traceable to hypertrophy of this kind is rare. I have repeatedly examined the urine of persons with immense enlargement of the left heart, without finding albumen in more than three or four instances. The following is one of these, in which no tenable explanation of the albuminuria could be found, except renal congestion from excessive power of the left ventricle.

T. H., æt. 21, a warehouseman, came under treatment Feb., 1864, suffering from immense cardiac hypertrophy. The apex beat in the seventh interspace, almost in the axillary line, and seven-and-a-half inches from the mid-sternal base. The impulse was strong; the whole body shook at each beat of the heart. The pulses were visible in all the superficial arteries. A loud to-and-fro, roughish murmur was heard over the aortic cartilage, of which the diastolic part was greatly prolonged. This murmur was heard loudly at the base, but grew weaker toward the apex—beyond which it ceased to be audible. The valvular mischief seemed to be confined to the aortic orifice. There was no sign of serious mitral regurgitation nor any indication of impediment on the right side of the heart. There was total absence of a cyanotic tint; on the contrary, the face was pinkish pale, and the margins of the lips and tongue were of a faint rose; there was no swelling of the veins of the neck nor a trace of anasarca. On the other hand the pulse was hard, resistent, bisferiens, ranging from 92 to 104. The character of the urine was highly significant. It was not high-coloured and scanty, as in venous congestion, but abundant, pale, and of low specific gravity. The daily discharge varied from 57 to 65 ounces, the spec. grav. from 1010 to 1015. It contained albumen, but only in small quantity; generally only a haze was produced with nitric acid; no tube-casts or other renal derivatives could be detected, though often looked for. It was distinctly observed that the proportion of albumen oscillated in accordance with the activity of the heart. When the ventricle was in high action, the albumen rose; when it became more quiescent, under the influence of rest and digitalis, the albumen almost vanished for a time. I have recently seen this patient again (March, 1865) and find that his state is still as above described; there is a trace of albumen in the urine; but the general condition is wonderfully good.

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* It is not probable that the *mean* lateral pressure in the arterial system can ever be raised above the normal degree in compensatory hypertrophy of the left ventricle; but it is quite conceivable and agreeable to clinical facts, that the *maximum* tension (attained at the close of the systole) may be excessive, and be counterbalanced, or more than counterbalanced, by undue diminution of tension during the ventricular diastole.

The treatment of active renal congestion will be described, with that of passive congestion, at the end of the next section.

PASSIVE CONGESTION.

The experiments of Robinson and Frerichs, already cited, show that an impediment or obstruction to the return of blood from the kidneys induces passive congestion of these organs, and, if sufficiently intense, causes albumen and blood to appear in the urine. An impediment of minor degree does not render the urine actually albuminous, but causes it to become scanty, high-coloured, dense, and prone to deposit abundance of lithates. Both these degrees of obstruction are frequently witnessed in clinical experience.

The obstruction may be seated in the chest, as in cases of valvular heart-disease, emphysema, and pleuritic effusion: or in the abdomen, as when a gravid uterus or other tumour presses upon the emulgent veins or the upper course of the inferior cava. Sometimes a cirrhotic liver compresses the latter vein as it lies in the hepatic fossa.

The alterations on the side of the urine are not always proportional to the degree of obstruction to the circulation. Cases are met with, in which venous stagnation exists in an intense degree, with dropsy, orthopnœa, and pulsating jugulars, without a trace of albumen in the urine; and others in which the urine-changes are strongly marked, while the more general symptoms of venous obstruction are only moderately so.

The two examples which follow, afford good illustrations of unusual degrees of renal derangement, secondary to obstructions to the circulation within the chest. In the first case the obstruction was due to old-standing tricuspid regurgitation; in the second to extensive emphysema.

Case I.—A lawyer's clerk, æt. 44, came under observation Dec. 6th, 1862. He was suffering from œdema of the legs, ascites, and a severe bronchial attack. The features were livid; but the veins of the neck were not distended. The heart's apex beat in the fifth interspace, a little outside the nipple line. The cardiac dulness extended four inches vertically, and about the same diagonally from base to apex. The heart's action was very irregular both in force and rhythm; pulse was 104. A loud blowing murmur was heard at the apex, of mitral regurgitant character, associated with a faint diastolic bruit, which was heard in maximum intensity over the second right costal cartilage. Loud bronchitic râles were heard universally over both lungs. There was copious mucous expectoration, sparsely speckled with blood; also severe dyspnœa, amounting at times to orthopnœa.

The urine was scanty, reddish, spec. grav. 1025, with abundant clouds of lithates. It contained a small quantity of albumen (equal to about ⅛). The deposit, examined under the microscope (see Fig. 39), revealed numerous scattered blood-disks; casts of tubes, mostly perfectly hyaline, sometimes only visible when tinted with magenta; some casts were dotted with withered renal epithelia or with the nuclei of these; no oily or fatty particles were found.

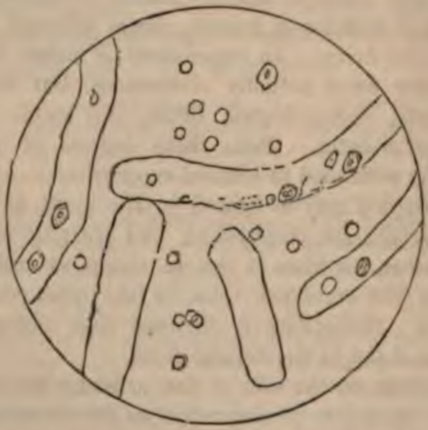

Fig. 39. Cast of tubes and blood corpuscles from the urine of a patient with passive renal congestion.

On tracing back the patient's history, it appeared that he had had five attacks of acute articular rheumatism, of which the earliest occurred in his twentieth year. In one of these the heart had become affected. The cyanotic appearance and dropsical symptoms had shown themselves some months previously, but had suddenly assumed a formidable intensity a fortnight before, in consequence of a bronchitic attack.

With rest and other appropriate means, the bronchial attack subsided in about ten days. The dropsical and dyspnœal symptoms receded, and a moderately quiescent state was attained. The urine underwent corresponding changes; it became more copious, its density fell, and the albumen faded to a mere trace: the casts remained as before.

While under observation this man went through three bronchitic attacks. In each, the urine went back to the character given of it in the first report. In the intervals again, the albumen became very scanty, and on two occasions the urine was found altogether free from albumen.

Case II.—On Oct. 16th, 1862, a strongly-built, stout woman, æt. 42, was admitted into the Manchester Infirmary, almost in a state of asphyxia. She was intensely blue in the face; could only breathe in the upright posture; her voice was a faint husky whisper; the tongue was livid; the veins of the neck were enormously dilated; she looked like a person half choked. There was not a particle of œdema nor any ascites; the limbs were firm and muscular. There was considerable drowsiness, but no actual coma. The sputum was frothy, not bloody. The examination of the chest revealed extensive capillary bronchitis, in emphysematous lungs; both bases were somewhat dull. There were no cardiac murmurs; the superficial cardiac dulness was inappreciable on account of the emphysema.

The urine was dusky red, and gave a play of colours with nitric acid (showing bile); no sugar in it; it was albuminous to a considerable degree (⅓). In the sediment, which was abundant and composed of lithates, were found numerous tube-casts—nearly all hyaline; some of them studded here and there with altered epithelium, or altered blood-disks; a number of free cells were also found, most of them pus corpuscles, but some with solitary nuclei—evidently renal, and very little altered from their natural appearance. One cast was seen so studded with these as to deserve the name of an epithelial cast. Not a particle of fat was found in the renal derivatives.

On the next day but one (Oct. 18), the patient seemed to breathe a little easier, but the surface was still intensely cyanotic. Of the urine, the notes state: "somewhat less highly coloured; very much less albuminous, in fact the urine only becomes hazy with nitric acid; casts and cells still abundant."

Next day the breathing seemed again, if anything, rather easier, but the strength was evidently failing, and the drowsiness was becoming deeper. The urine no longer showed any bile tints, though still of a deep brown colour. Albumen could only be discovered in it by very careful testing; the casts had all but disappeared; a few short fragments (slightly more granular than before) could with difficulty be found and identified. A few blood corpuscles were seen after diligent searching.

Oct. 20.—There was evident emaciation going on, and steady diminution of strength. Scarcely any nourishment had been taken since admission; the voice was whispering, and the surface livid. The dyspnœal symptoms were at a stand-still; drowsiness on the increase.

Oct. 21.—The urine was now quite free from albumen, and no casts could be found in the deposit. In the course of the succeeding night the patient quietly died, as if in sleep, partly exhausted by want of nourishment and the efforts to breathe, partly poisoned by the mephitic condition of the blood.

Autopsy, 18 hours after death. The *heart* weighed eleven ounces; the valves were healthy; a few slight atheromatous patches existed in the aorta. The *lungs* were in a state of excessive and universal emphysema; they bellied out of their cavities, when the chest was opened, like bladders of air. Spots of intense congestion were found here and there on section, and the extreme bases were somewhat œdematous. The *liver* was enlarged and congested. The *kidneys* were considerably enlarged, and weighed together twelve ounces; the capsules peeled off readily. On section the pyramidal and cortical substances were distinct from each other, and in due proportion; both parts were intensely congested, but otherwise natural, both to the naked eye and to microscopical examination. The body was still moderately well-nourished; there was no anasarca in any part, nor any ascites.

There is one circumstance in this history which at first sight appears contradictory, namely, the disappearance of the albumen from the urine, notwithstanding that the obstruction in the chest persisted or even increased, and, indeed, brought the circulation ultimately to a stand-still. The explanation of this occurrence is, I believe, to be found in the diminishing pressure in the arterial system from the gradual failure of the heart's power. Some of Robinson's experiments bear clearly on this point. He found, on ligaturing the renal veins in rabbits, that *vigorous* animals exhibited the urine-changes (albuminuria, &c.) in far greater intensity than *weaker* animals; and he attributed the difference to the fact that in strong animals the powerful contractions of the ventricle served to maintain a greater counter-pressure on the arterial side of the renal circulation, and in this way intensified the intrarenal pressure or congestion. In the patient before us the pressure on the arterial side was visibly declining from day to day, in consequence of the inability to take food which diminished the mass of the blood, and the progressive poisoning of the blood (from defective respiration) which gradually depressed, and finally annihilated, the contractility of the ventricle.

The state of the kidneys in passive congestion varies with the duration of the obstruction. When the obstruction has been only recently established, as in the woman whose case has just been related, the kidneys are found simply enlarged and engorged; they resemble the kidneys of the rabbits whose renal veins were ligatured by Robinson. But when the congestion has been in existence for months and years the kidneys are found to have undergone far more profound alterations.

The exact nature of the alterations in this latter case has been a matter of dispute. Frerichs, Bergson, and Bamberger consider them as identical with those in the granular kidney of Bright's disease; but Traube contends that they are essentially different. The following description of the kidneys, in old standing cases of passive renal congestion, is based on a comparison of several accurate examinations by the above-named authors. The organs are uniformly reddened; they are decidedly smaller and harder than natural; their surface is generally smooth, sometimes granular; the proportion of cortical to pyramidal substance is not much altered; in very chronic cases the cortex may be somewhat atrophied. Rosenstein describes the epithelium of the convoluted tubes as irregular in shape, and containing granular matter which is sometimes fatty. The straight tubes are often dilated, varicose, and filled with granular, opaque epithelium. The basement membrane of the canals is thickened, and the venous radicles greatly dilated. The Malpighian bodies are highly injected, but otherwise intact; in very protracted cases they may become a little atrophied, and their capsules thickened. The intertubular matrix of connective tissue is increased in quantity, especially in the pyramidal portions.

Whatever anatomical difficulties there may be in the way of separating these cases from chronic Bright's disease, the clinical distinctions between them are clear and undoubted. Renal disorder from passive congestion comports itself quite differently from Bright's disease of independent origin, and from Bright's disease coming on in the course of chronic disease of bone or phthisis. The contrast between the first and the third is very marked. In the first (passive congestion), the renal affection has no momentum of its own, and makes no independent progress; it oscillates with the rising and falling intensity of the venous obstruction; it remains throughout a subsidiary complication of the primary disease, and assumes none of the special characteristics of Bright's disease (uræmia, &c.). On the other hand, when renal disease declares itself in the course of chronic phthisis, it assumes at once a formidable position. The entire clinical complexion of the case is transformed. Sometimes even, the pulmonary disorder is altogether supplanted and thrown into the background by the more rapid progress and fatal course of the renal disease (see case of M. C. in chap. iv.).

Treatment of Congestion of the Kidneys.—Congestion of the kidneys, whether active or passive, does not often call for separate treatment. Its course and intensity are usually contingent on the progress of the primary disorder of which it is a secondary phenomenon. But sometimes active congestion has an independent origin; in other cases, although secondary, it is sufficiently threatening to demand special attention. Passive congestion from cardiac and pulmonary obstructions can be most efficiently relieved by remedies applied to the primary complaints. But passive congestion from the pressure of a pregnant uterus—cases which will be considered at length in the appendix to this chapter—not unfrequently claims energetic treatment on its own account.

The most efficient means of combating active renal congestion are complete rest of the body, cupping the loins, brisk purgatives, the warm bath, and other diaphoretics. In the passive cases, cupping can only be of service when the congestion is due to a temporary cause, such as pregnancy; in the more common cases, the application of gentle counter-irritants to the loins is more serviceable, namely, tincture of iodine, embrocations, &c. Derivation by the bowels and skin is also an important means of relieving the overloaded organs.

APPENDIX.

On the connection of renal congestion, albuminuria, and Bright's disease, with pregnancy and puerperal eclampsia.

LEVER—Guy's Hospital Reports, 1843, p. 495.
DEVILLIERS and REGNAULD—Archives Générales, 1848.
FRERICHS, l. c., p. 211.
WIEGER—Schmidt's Jahrbücher, Band 87, p. 57.
BRAUN—On uræmic convulsions in pregnancy and parturition. Translated by Dr. Matthews Duncan. Edin. 1857.
ROSENSTEIN, l. c., p. 52.
ABEILLE—Traité des Maladies à urines albumineuses et sucrées. Paris, 1863.

Professor Simpson was, I believe, the first to call attention to the occasional presence of albumen in the urine of pregnant women. The subject has since been studied on many hands, with a view to elucidate the connection of the puerperal state, and especially of puerperal convulsions, with Bright's disease.

Albuminuria does not usually show itself in pregnancy until the seventh or eighth month, and often not until the approach of labour. Sometimes, however, it appears earlier, even so early as the third month. It is generally attended with œdema of the lower extremities, sometimes also of the face and upper parts of the body.

Blot found among the patients of a lying-in hospital, that one pregnant woman in five had albumen in the urine: this estimate is evidently very much too high for a general average. Abeille found, in private practice, the proportion to be one in ten; and Van Arsdale and Elliott, one in fifty-six.—(New York Journ. of Med., 1856). This last estimate is probably the most nearly correct.

The albuminuria of pregnancy, and the accompanying anasarca, usually go on increasing up to the time of delivery, and then rapidly pass away. As a rule, the albumen is quite gone in forty-eight hours, sometimes even in twenty-four hours; but it may not wholly disappear for ten or fifteen days. If it continue beyond this last period, the gravest apprehensions of organic renal disease are justified.

A certain number of pregnant women having albuminuria (probably about one in ten) are affected with epileptiform convulsions (or eclampsia) before, during, or after labour.*

If we inquire into the origin of albuminuria in pregnancy, two conditions present themselves, which, together or separately, are capable of explaining its occurrence: these are (*a*) passive congestion of the kidneys from pressure of the womb on the renal veins, and (*b*) the altered quality of the blood which is proper to the pregnant state.

The growing womb, mounting into the abdomen, necessarily exercises a certain compression on the contents of that cavity; and, among other structures, on the inferior cava and renal veins. This mechanical pressure occasions a passive congestion of the kidneys, which, if sufficiently severe, induces albumen to appear in the urine, together with blood and tube-casts. That this is one of the most efficient causes of albuminuria in pregnancy, is indicated by the fact that primiparæ, in whom the parts are resistent, and the pressure therefore intense, are disproportionately liable to albuminuria; also by the fact that albumen does not usually appear in the urine until the later periods of gestation, when the venous stagnation has reached its height.

The altered condition of the blood probably contributes more to the establishment of the œdematous swellings, especially in the upper parts of the body, than the mechanical pressure. The blood, in pregnancy, is poorer in red corpuscles and more watery than natural—a condition highly favourable to serous transudation, and to the production of anasarca and albuminous urine.

The expulsion of the fœtus is, as has been stated, commonly the signal for the disappearance of the œdematous swellings, and the restoration of the urine to its healthy state; but in a certain number of cases eclamptic convulsions break out about the time of parturition, and of these about 30 per cent. prove fatal; in certain other cases the albuminuria does not disappear after delivery, but persists, with or without dropsical effusions, until there is no longer any doubt that genuine and confirmed Bright's disease has been established.

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* The liability of albuminuric pregnant women to eclampsia, is estimated at a much higher rate than this by Blot, Mayer, and Devilliers and Regnauld. The united statistics of these observers gives a proportion of about 1 in 4. Taking all pregnancies together—with and without albuminuria—about 1 in 500 are complicated with eclampsia. Braun gives the proportion as 1 in 545.

There has been much dispute as to the exact nature of the connection between the events here enumerated and the puerperal state. It has been on the one hand alleged, and on the other hand denied, that the pregnant state is an effective exciting cause of Bright's disease: it has also been both alleged and denied that puerperal convulsions are of renal (uræmic) origin.

There can be no doubt that many of the cases in which Bright's disease co-exists with, or follows, the pregnant state, are examples of the coincidence of two mutually independent conditions. Pregnant women are of course liable, like other persons, to contract Bright's disease from any of its ordinary causes; and, again, women who are already the subjects of Bright's disease may become pregnant. But after eliminating the cases belonging to these two categories, there are still, as I believe, a considerable number in which Bright's disease has been really *caused* by pregnancy. The Registrar-General's reports furnish some valuable evidence on this point. In the five years 1857—61, there were registered 6220 deaths from Bright's disease. Of these 3699 were males, and 2521 females —being in the proportion of 68 females to every 100 males: this was the relative proportion between the two sexes at all ages. But the deaths of women from Bright's disease during the child-bearing years of life (from twenty to forty-five), far exceeded this proportion—being as high as 80 women to every 100 men. After the age of 45, the proportion of deaths from Bright's disease sank again to 59 women for every 100 men. There seems no other conclusion to be drawn from these numbers, than that the puerperal state is a prolific cause of Bright's disease.

Another question of considerable interest is whether puerperal eclampsia is due, or not, to uræmic poisoning. The affirmative has been warmly supported by Frerichs, Braun, Litzmann, Wieger and others; and the negative by Scanzoni, Depaul, Rosenstein, and several more.

On the affirmative side it has been shown:—(1) that eclamptic fits are similar, symptomatically, to uræmic convulsions; (2) that albumen is almost invariably present in the urine of eclamptic patients; and that in several cases undoubted evidence of Bright's disease has been found in the kidneys after death; (3) that, frequently, anasarca of the upper parts of the body, and dryness of the skin, coexist with albuminuria, and confirm the diagnosis of Bright's disease.

On the opposite side it is alleged:—(1) that there are authentic instances (apart from epilepsy, apoplexy, or hysteria) of puerperal eclampsia without albuminuria;* (2) that anatomical evidence of Bright's disease has only been found in a minority of the cases; that more frequently the kidneys have only been found congested, without any organic alterations which could be identified with any form of Bright's disease; (3) that other causes (than Bright's disease) have been repeatedly found in the bodies of persons dying of puerperal eclampsia, namely, œdema of the brain, and congestion of the meninges, which probably were not without concern in bringing about the attacks.

The want of segregation of irrelevant cases prevents the possibility of a clear analysis of the facts adduced in this dispute. But it is evident that the existence of well-attested cases of eclampsia without a trace of albumen in the urine, is fatal to the *universality* of the uræmic theory. On the other hand, the not infrequent co-existence of undoubted Bright's disease, leads strongly to the conviction, that, in many cases, the convulsions are truly uræmic. It must not, however, be forgotten, that pregnant women who are the subjects of confirmed Bright's disease frequently pass through labour without the least convulsive disturbance.

As the evidence now stands, puerperal eclampsia cannot always be attributed to one and the same invariable cause. In some instances, the convulsions appear to be essentially of a reflex character, arising from irritation of the generative organs, acting on a nervous system in a state of exalted sensibility. It is at the period when this sensibility attains its maximum, namely, during the act of labour, that convulsions usually break forth. But it is likewise about the same period that the pressure within the abdomen becomes most intense, and the stagnation in the renal veins and interruption to the secretion of urine most complete. When the act of birth commences there are added to these causes of disturbance, violent and general muscular contractions causing suffusion of the features and congestion of the cephalic meninges. Several explosive elements are thus brought together at the same period; and it is scarcely to be wondered at, that the equilibrium of the nervous system should be thereby occasionally overset.

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* Among other examples, the following may be referred to:—Abeille, l. c., p. 607; Riedel, Zeitschr. f. d. Geburtsheilk., 1858, p. 13; Rossi, ibid. 1863, Bd. II., p. 72.

The recognition of two or more categories of puerperal eclampsia, is of much importance both for prognosis and treatment; and the want of some rational classification of the cases is doubtless one cause of the discrepancies in the experience of different observers as to the beneficial effects of venesection and other plans of treatment.

At least three categories seem to deserve to be recognised, viz., 1. *Cases depending on confirmed and chronic Bright's disease.* In these the eclampsia must be regarded as mainly or wholly uræmic; the ultimate prognosis is lethal, and depletive measures are less indicated than chloroform, etc. 2. *Cases depending on passive congestion of the kidneys, or on a condition resembling, if not identical with, acute Bright's disease.* These are usually primiparæ; the phenomena are, probably, partly uræmic, and partly reflex; the prognosis is favourable, were the fits once over; active depletive measures are indicated. 3. *Cases depending on reflected uterine irritation and meningeal congestion.* In these the urine is *not* albuminous; the prognosis is favourable, were the fits over; they call for active depletory measures.

Something remains to be said in the way of diagnostic indication, in cases of pregnancy complicated with albuminuria.

The urine of a pregnant woman being found albuminous—how shall it be known, whether there exists confirmed Bright's disease or only a temporary disorder which will harmlessly subside after parturition? The following points tell strongly for confirmed Bright's disease—an abundant flow of pale urine of low density; presence of granular or fatty casts; a considerable amount of albumen and yet a relaxed state of the abdomen and tissues generally; anæmia; a hypertrophied left ventricle ; anasarca equally distributed (or nearly so) over the whole body. The points which, on the other hand, tell in favour of congestion, or of acute (and curable) Bright's disease are—evident signs of severe pressure within the abdomen; the patient being a primipara; the quantity of albumen in the urine appearing to bear a proportion to the existing venous congestion; the urine being high-coloured, scanty, and dense ; the anasarca being mostly, or altogether, confined to the lower extremities ; absence of anæmia and cardiac hypertrophy.

By attention to these points I have been able, in the cases which have fallen under my observation, to frame a diagnosis which the event has justified.

CHAPTER II.

BRIGHT'S DISEASE.

Bright—Reports of medical cases, vol. i. Lond. 1827. Also papers in Guy's Hospital Reports for 1836 and 1840.

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Osborne—On dropsies connected with suppressed perspiration and coagulable urine. Lond. 1835.

Rayer—Traité des Maladies des Reins. Paris, 1839, 1840.

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Frerichs—Die Bright'sche Nierenkrankheit. Braunschweig, 1851.

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Zalesky—Untersuch. ü. d. urämischen Process. Tub., 1865.

PRELIMINARY REMARKS.

CASES characterized by albuminuria and dropsy, depending on structural changes in the kidneys, are classed together under the general title of BRIGHT'S DISEASE.

Several different pathological states are doubtless included under this designation; and the cases present considerable diversity, not only in the acuteness of their course but also in their modes of origin and symptoms. Numerous attempts have been made, to divide and classify the various conditions of the kidney found after death from Bright's disease; and to connect each with its appropriate clinical history. Hitherto none of these attempts have obtained general assent; and a regrettable confusion of nomenclature has been added to the inherent intricacies of the subject. Nothwithstanding the diversities just referred to, the points of resemblance between the several varieties of Bright's disease, are so strong and so numerous, that they form an easily recognised clinical group. This resemblance arises, in great part, from the circumstance that the structural changes in the kidneys, various as they may be, bring about the same ultimate results, namely, impoverishment of the blood from loss of albumen, with poisoning of it from retention within the body of the excrementitious matters of the urine; and the more prominent symptoms in Bright's disease, arise from this changed condition of the blood, rather than from the *direct* effects of the structural changes in the kidneys.

Opinions are divided, in the first place, as to whether there be a fundamental unity beneath the apparent diversity; in other words, whether the "large, smooth, white kidney," the "small smooth kidney," the "granular uncontracted kidney," and the "granular contracted kidney," are successive stages of one and the same pathological process, or represent radically distinct diseases.

Dr. Bright, whose researches on this subject have made his name so renowned in medical science, expresses himself quite doubtfully on this point. In his introductory remarks to the twenty-three cases first published by him in 1827, he says :—

"From the observations which I have made, I have been led to believe that there may be several forms of disease to which the kidney becomes liable in the progress of dropsical affections. I have even thought that the organic derangements which have already presented themselves to my notice, will authorise the establishment of *three* varieties, if not of three completely separate forms of diseased structure." But toward the close of the same remarks he observes :—"Although I hazard a conjecture as to the existence of these three different forms of disease, I am by no means confident of the correctness of this view. On the contrary, it may be that the first form of degeneracy to which I refer never goes much beyond the first stage; and that all the other cases, together with the second series and the third, are to be considered only as modifications, and more or less advanced states of one and the same disease."—(Reports, pp. 67 and 69.)

Soon after the period when these sentences were written, a new vantage ground for the study of renal diseases was acquired by the researches of Mr. Bowman, which threw a strong light on the intricate anatomy of the kidney. Histologists of eminence both in this country and Germany—Busk, Toynbee, Simon, Henle, Rokitansky, Virchow—and inquirers who have made the subject a special study—Johnson, Frerichs, Basham, Dickinson, and many more—have worked with unexampled perseverance to ascertain the nature and arrange the varieties of the morbid processes taking place in the kidneys in Bright's disease; and yet the same doubt which possessed the mind of Bright hangs over the subject at the present day.

All this labour has not, of course, been thrown away. On the contrary, much light has been shed on a number of points; and data of importance have been obtained for prognosis and treatment. More especially the examination of the organic admixtures of the urine—renal epithelium and casts of the uriniferous tubes—has yielded to Dr. Geo. Johnson results of the highest clinical value, which claim for him a pre-eminent mention in this field of pathology.

Frerichs considers that Bright's disease is essentially one, and that it is of an inflammatory nature. He divides the anatomical changes in the kidneys into three forms, which he regards as stages of the same fundamental process, namely:—

1. The stage of hyperæmia and commencing exudation.
2. The stage of exudation and commencing change of the exudation.
3. The stage of degeneration and atrophy.

Dr. Johnson, on the other hand, recognises several distinct processes under the common heading of Bright's disease—but chiefly two, and both of an inflammatory nature—one characterized by a shedding and destruction of the epithelial lining of the uriniferous tubes (*desquamative nephritis*), and one without such desquamation, and affecting the intertubular structures of the organ (*non-desquamative nephritis*). He also gives a separate place to "fatty degeneration" and "waxy degeneration" of the kidney.

Dr. Dickinson, in two remarkable papers, based on the examination of the kidneys and the clinical records of 369 cases which terminated fatally in St. George's Hospital, divides the disease into two varieties :—1. *Tubular disease*, which consists in a catarrhal affection of the uriniferous tubes. In this variety, the kidney is smooth on the surface, enlarged, and red or white in colour; in very advanced cases (which are rare) the kidney becomes atrophied : 2. *Intertubular disease*, in which he conceives that an exudation is thrown out, and penetrates between the tubuli, involving them and the Malpighian bodies in destruction. In this second variety the kidney is always granulated on the surface, and, very generally, atrophied.

It would lead me too far to discuss the merits of these and the many other classifications which have been put forth. I content myself with simply indicating the more important ones. In the following pages the subject will be treated from a clinical, rather than an anatomical, point of view, and the cases will be classified under the two main heads of *acute* and *chronic* Bright's disease. The former embraces a compact and universally recognised group, which formerly went under the designation of "inflammatory dropsy." It corresponds to the acute desquamative nephritis of Johnson, to the first stage of Frerichs, and to the acute tubular disease of Dickinson. The latter includes the protracted cases, which either have lapsed into a chronic state from the acute form, or, which is far more frequent, have been chronic from the beginning. Three types of chronic Bright's disease will be recognised—1. Cases which have lapsed from the acute state (kidney smooth, white, generally large, exceptionally dwindled). 2. Cases which have been chronic from the beginning (kidney granular, red, contracting). 3. Cases associated with waxy or lardaceous (so-called amyloid) degeneration of the kidneys.

The presence of fat in the renal substance, and in the epithelium of the tubes, is not special to any one type of renal degeneration; but is found associated with anatomical changes of the most varied kinds: it has therefore no claim to a separate consideration.*

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* NOTE ON THE RECENT ADVANCES IN THE ANATOMY OF THE KIDNEY.—Much reliance cannot be placed on the exactness of the descriptions hitherto made of the microscopical changes occurring in the kidneys in Bright's disease. The flagrant discrepancies between the descriptions of various high authorities is notorious. It is for this reason that I have adopted a clinical instead of an anatomical basis of classification; and that in the account of the morbid anatomy of the disease, greater prominence is given to the alterations appreciable by the naked eye than to those which are revealed by the microscope. The explanation of many of the discrepancies here alluded to is, doubtless, to be found in the erroneous views hitherto held respecting the normal anatomy of the kidney.

The recent researches of Henle, Luschka, Krause, Chrzonszczewsky, Colberg, Ludwig and Zawarykin, Roth, Schweigger-Seidel and other German inquirers, make it abundantly evident, that the account hitherto accepted, on the authority of Mr. Bowman, of the course and structure of the uriniferous tubes, requires considerable modification. Henle, in the remarkable brochure which, in 1862, gave a new impulse to this inquiry, announced that there existed in the pyramidal portions of the kidney two essentially different orders of tubes. The one order consisted of larger tubes, which ran downwards to open on the papillæ; these he called the *open* canals. The other order consisted of much narrower tubes, which, after running down a varying distance into the pyramids, made a sharp loop-like bend, and reascended into the cortical substance; these latter he named *looped* canals. Henle believed that these two systems of tubes were, throughout, distinct from each other; that the open canals, starting from the openings on the papillæ, ascended into the cortex, and then divided and subdivided until at length they formed a network of communicating channels; that the looped canals, on the other hand, constituted a closed system of tubes, each thread of which began in a Malpighian body, and, after sundry convolutions, descended into a pyramid, where it turned on itself in the form of a loop, reascended into the cortex, and finally terminated in a second Malpighian body.

The subsequent researches of Ludwig and Zawarykin, of Roth, and of Schweigger-Seidel—made independently of each other, and remarkably in agreement on the main points—show that Henle was in error in supposing that the two systems of canals did not communicate with each other. These observers succeeded in establishing the continuity of the tubes which originate

GENERAL ETIOLOGY OF BRIGHT'S DISEASE.

THE special etiology of the several types of Bright's disease will be separately considered in the two following chapters, but it will be convenient in this place to consider some of the points bearing on the etiology of Bright's disease as a whole.

The want of uniformity in our nomenclature of organic diseases of the kidneys has considerably lessened the value of the returns of the Registrar-General in this field of pathology. Cases registered on the certificate of death as "Bright's disease" are entered in these returns as "nephria;" but it is evident that the larger number, even of the cases recognised as Bright's

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in the Malpighian bodies, with the excretory canals which debouch into the infundibula on the surface of the papillæ—in so far restoring the original view of Bowman. It cannot be said that the termination of the inquiry has yet been reached; the subject is still *sub judice;* but enough has been done to render it highly probable that the following scheme (after Schweigger-Seidel) of the course and structure of the uriniferous canals is, in the main, correct.

If the course of a uriniferous tube be followed in the direction taken by the urine, we shall find that, starting from a Malpighian body, it immediately falls into a knot of windings or convolutions. Emerging from these, it bends downwards towards the pyramids, and following a straight course, descends into one of these to a varying depth; it then turns up again, and reascends into the cortical part; here it forms a second and smaller series of windings, and finally bends down once more, joins with others similar to itself, and passes straight into the pyramidal portion, when it unites, two and two, with other straight canals, and issues at length, greatly enlarged, in an open mouth on the surface of a papilla.

The looped section of the tube, which dips into the pyramids (that portion which intervenes between the two knots of windings) undergoes, in a considerable length of its course, a remarkable narrowing of its diameter. This narrow part is only about one-third of the width of the remainder of the canal. The narrowing is due to a change in the epithelium, which, in this part, consists of perfectly clear small flat cells, with prominent nuclei, whereas in the wider portions, the epithelium is composed of thick opaque granular cells which nearly fill the lumen of the tube. The available bore of the narrow part appears to be as large as that of the wider portions. The narrow part generally forms the actual bend of the loop, and a certain length of its descending and ascending limb; but sometimes the narrowing begins and comes to an end in the descending limb of the loop. The following references may be consulted for full details on this subject:—Henle—*Zur Anatomie der Niere.* Gött., 1862; Luschka—*Anatomie des Menschen.* Tubingen, 1863, p. 299; Ludwig and Zawarykin—*Sitzungsb. d. Kais. Akad. der Wissenschaften.* Vienna, 1863; Roth—*Schweizer. Zeitschr. f. Heilk.* Bern., 1864, p. 1; Chrzonszczewsky—*Archiv f. path. Anat.*, 1864, p. 153; Schweigger-Seidel—*Die Nieren des Menschen.* Halle, 1865; Frey—*Das Microscop,* &c., p. 361. Leipz., 1863; also the new edition of Henle's Anatomy.

disease during life, are not so registered, but are classified under the heads "nephritis" and "kidney disease." To obtain some idea of the prevalence of Bright's disease, let us take the numbers under these three designations. There were registered in England and Wales, in 1861 :—

1448	deaths from	"nephria,"
306	"	"nephritis,"
2318	"	"kidney disease,"

—making a total of 4072. This yields only a proportion of 0·93 per cent. of the total deaths from all causes—a number which is probably considerably below the true proportionate mortality from Bright's disease. Without admitting, with Mr. Simon, that two-thirds of the cases of Bright's disease run a latent or undiscovered course, it must be allowed that a very large number are overlooked in these returns, and are probably to be found among the 7301 entered as "dropsy," or among those entered under "convulsions," "pneumonia," and other headings.*

Bright's disease is about one-third more common among men than women (863 males to 585 females). This excess of deaths among males, although present at every age, is not equal at the different periods of life: it is most marked between the ages of forty-five and sixty-five.

The mortality from Bright's disease shows a progressive increase from childhood up to the age of 45; in the succeeding 20 years (45 to 65) the mortality continues steady, at a somewhat lower, but still high, rate; the next decade (65-75) shows a decided diminution as regards Bright's disease, though the general mortality at this epoch is at its highest point. These facts are exhibited in the table on the following page :—

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* It is evident, however, that Bright's disease is gradually becoming better known in this country, and more frequently identified. In 1852, only 570 deaths were entered under "Nephria." In each successive year the number rose, quite out of all proportion to the increase of the population, until in 1861 it reached 1448, nearly thrice as many as in 1852. Correspondingly, the entries under "dropsy" diminished, from 9788 to 7301, for the same two years.

TABLE *showing the number of Deaths registered as "Nephria" (Bright's disease) in England and Wales, in* 1861, *at the different periods of life* :—

	Under 5 yrs.	5—15 yrs.	15—25 yrs.	25—35 yrs.	35—45 yrs.	45—55 yrs.	55—65 yrs.	65—75 yrs.	75 years and upwds.	Total at all ages.
Males .	33	45	64	114	171	159	146	104	27	863
Females .	27	34	61	84	116	87	98	60	18	585
Both sexes	60	79	125	198	287	246	244	164	45	1448

That complex of impressions which is familiarly known as taking *cold* is the common cause of Bright's disease in its acute form. *Cold*, operating more slowly and continuously, also constitutes a prolific source of chronic Bright's disease. Persons whose occupation exposes them to cold, wet, and the inclemencies of the seasons, without adequate protection—those who work in hot workshops, and are in the habit of going to cool their reeking bodies in the open air—the indigent classes, who dwell in damp cellars, insufficiently clad and ill-fed, amid dirt and squalor, furnish a large quota of victims to this disease.

Dr. Johnson is at especial pains to explain the *modus operandi* of this frequent cause of renal disease. He contends that the defective action of the skin causes certain deleterious matters to accumulate in the blood, and that the burden of their elimination is thrown upon the kidneys, which receive injury thereby. It is impossible to accept this view without great limitation, seeing that suppressed cutaneous transpiration ushers-in a multitude of inflammatory and febrile conditions, without provoking renal disease. When a person "takes cold," it is a fact, that, the secretion of the skin is very much diminished or altogether suppressed: but it is not possible to predicate on what organ the injurious impression will ultimately settle—whether on the bronchial tubes, the pleura, the lung-tissue, the kidneys, or some other organ or part of the body; so that it cannot be maintained that there is any *special* relation between suppressed cutaneous secretion and the genesis of renal disease.

The abuse of spirituous liquors ranks high—probably higher than any other single circumstance—as a determining cause of Bright's disease. Christison estimates the proportion due to this cause, in Edinburgh, as three-fourths or four-fifths of all the cases; and he justly remarks that it is not habitual drunkards only who show this tendency to renal disease, but dram-drinkers, who are in the constant practice of using ardent spirits several times in the course of the day, without becoming actually intoxicated. In this neighbourhood the cases due to intemperance have not seemed to me to exceed a fourth of the whole.

Malt liquors—though far less pernicious than spirits—are not without influence to produce Bright's disease when largely indulged in. In a journeyman baker, recently under my care at the Infirmary, the disease was clearly traced to a habit of fuddling himself with beer from Saturday evening to Monday morning, which the patient had followed for several years.

Very frequently, intemperate habits go hand in hand with a grimy skin and exposed occupations; and the subjects of Bright's disease are found disproportionately numerous among labourers, well-sinkers, cabmen, carters, hawkers, glass-blowers, smelters, and puddlers.

A large number of cases arise in connection with some *constitutional vice*, more especially tuberculosis or struma. Among the easier classes, gout and constitutional syphilis are prominent antecedents.

I have not met with any case occurring as a sequela to *ague*. Both Becquerel and Frerichs likewise state that, in dropsies following intermittent fevers, they had never found evidence of kidney disease; but Rosenstein found no less than 23 per cent. of the cases of Bright's disease in the Dantzic Hospital referrible to antecedent ague. The character of the epidemic (as with scarlatina) exercises a marked influence on the frequency of this complication. Heidenhain, who had an opportunity of watching a series of epidemics of ague in Marienwerder, states that neither dropsy nor renal mischief occurred in the earlier ones; but in the latest epidemic secondary renal disease was encountered in almost every case.*

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* Rosenstein, l. c., p. 209.

Chronic affections of the lower urinary passages (cystitis, stricture, etc.) frequently lay the foundations of renal disease. In a boy of seven, who lately died in the Royal Infirmary, a small stone no larger than an almond was found lodged near the neck of the bladder. Repeated sounding had failed to detect it during life; operation was consequently not performed. For some weeks before death, general anasarca had shown itself. The kidneys were found wasted to an extreme degree; the cortical substance was reduced to a thin edge no thicker than a shilling; the pelvis of the kidney and the ureters were dilated, and their lining membrane thickened and bathed in pus. In cases of this class there is a double influence tending to produce renal degeneration, namely, the long continued exhausting suppuration and direct transmission of the inflammatory process by continuity of tissue.

The use of mercury, which Wells and Blackall believed capable of producing albuminuria and renal mischief, has not been found, by observers of wider experience, to have this effect. Rayer and Desir, out of forty cases treated with mercury at the Hôpital des Vénériens, only found a slight quantity of albumen in two—in both of which its presence was accounted for by the existence of pus in the urine. Rayer further observes that he had for years used a multitude of mercurial preparations in the treatment of various diseases, without ever having observed the production of dropsy. He also states that he had treated a large number of gilders affected with mercurial trembling, and that he had not seen a single case of dropsy with coagulable urine supervene during or after this trembling (see also p. 121, note).

A certain number of cases of chronic Bright's disease present themselves, in which the most searching analysis fail to indicate the exciting cause of the disorder. In some of these the renal affection is only a part-manifestation of some wide-spread cachexy, as in an example (M. H.) to be related in chap. iv., in which fatty degeneration co-existed in the heart, great vessels, brain, and kidneys.

CHAPTER III.

ACUTE BRIGHT'S DISEASE.

(Acute Diffuse Nephritis.)

See references prefixed to Chap. II., p. 293.

Anatomical characters.—The kidneys are always more or less enlarged—sometimes to twice their natural size; their surface is smooth; the capsule thin, transparent, and easily stripped off; their colour varies; it is generally a deep dusky red; but sometimes a light fawn, almost white; in other cases it is mottled red and white. The superficial veins are larger and more distinct than natural. When the kidney is cut open the cortical substance is found to be increased very much out of proportion to the pyramidal. The *red* congested kidney exudes a bloody sanies abundantly from the cut surface; and a number of hæmorrhagic spots may be generally seen scattered through the cortex or beneath the capsule. The surface of the section is dusky red, and studded with minute darker-red points, which Rayer thought to be engorged Malpighian corpuscles, but which Mr. Bowman showed to be loops or knots of convoluted uriniferous tubes distended with blood. The *pale* and the *mottled* kidneys present a contrast of colour between the cortex and the pyramids. The latter appear unnaturally red, and from their bases radiating lines of red spread, fan-like, into the cortical substance. The cortical portion is smooth and white, or yellowish-white, and spotted like ivory.

Under the microscope, the chief, and characteristic, alteration is found to be, an immense increase of the epithelial contents of the convoluted tubes. The individual cells may be natural in appearance; more commonly they are granular, opaque, or disintegrated; and sometimes a few oily particles are detected in them. The lumen of the tubules is choked up with epithelium; and, in extreme examples, their diameters are increased to twice and even thrice their normal measurements. Mixed with the epithelium is more or less blood. In the straight tubes evidence of the same process is seen, in a less intense degree. Their larger bore and direct course favours the escape of the detached epithelium, so that some of them are partially or wholly denuded. Fibrinous exudation is found in the tubes of the cortical and pyramidal portions, forming glassy cylinders, of various size, according as the tubes have preserved or have shed their proper lining.

The renal capillaries and the Malpighian tufts are either intensely injected, or they preserve their natural appearance. The intertubular matrix is unaffected.

The morbid process seems to consist, essentially, in a catarrhal condition of the uriniferous tubes, with a prodigious proliferation of their epithelial elements. At the first, there is an inflammatory congestion of the organs, with rapid swelling, and more or less extensive rupture of the capillaries, especially those of the Malpighian bodies. On that follows increased production of epithelial cells: these multiply, choke up and distend the uriniferous tubes, thereby compressing the renal capillaries and impeding the circulation through them. When this proliferation has reached a certain degree, the kidneys, which before were of a dusky red, become pale or mottled—not so much from an actual deficiency of blood in the organs, but rather, as Dickinson explains, from the white colour of the masses of epithelium overpowering the natural red of the parts.

The choking-up of the tubes with their own secretion necessarily impedes the depurating functions of the kidneys, and the blood is poisoned with excrementitious matters. The urine becomes scanty in amount, and deficient in its proper constituents; it carries with it, as it percolates the diseased ducts, loose epithelium, blood and fibrinous exudation, or detaches whole tracts of the lining, all of which objects form an abundant grumous sediment in the urine.

How soon the change from red to white takes place, depends on the rapidity of the multiplication of the epithelial cells. I have seen the bloodless condition reach an extreme degree in six weeks. Dr. Dickinson states that it may occur within four days.

Course and symptoms.—The invasion of the disease is commonly abrupt, and traceable to some definite cause. A person takes cold, or falls into a fit of intemperance, and next morning, or in two or three days, the face begins to swell, then the hands and body generally. In another large class of cases the disease breaks out during convalescence from scarlet fever or—but much less frequently—some other febrile or zymotic complaint.

Acute Bright's disease is usually ushered in with chilliness or shivering, headache, nausea, vomiting, pains in the back and limbs, arrest of the cutaneous perspiration and oppression in the chest. When fairly established, the symptoms are exceedingly distinctive. The countenance is pale and puffy, with a heavy stupid expression; the limbs and trunk are anasarcous. The œdematous parts are resistent on pressure, and pit little or none. More or less effusion takes place into the serous cavities, especially the pleura and peritoneum.

There is a general febrile movement; the pulse is hard and full, the appetite lost, thirst excessive; the skin is dry, and the whole surface blanched and tumefied. An uneasiness or dull pain is felt in the loins, and the renal regions are tender on pressure.

The *urine* is of a smoky or dusky hue—in some instances dark brown like porter—from the presence of altered blood. On standing, it deposits a copious, flocculent, dirty-brown or chocolate sediment, like the settling from beef-tea. It is very albuminous; it may even become quite solid on boiling. The specific gravity, in the stage of increment, is usually above 1020, often much higher, mounting sometimes to 1030, and in one instance which occurred to me even to 1065. When of high density, the urine is proportionally scanty; it may not exceed 12 or 18 ounces in the twenty-four hours; in extreme cases it may sink to 6 ounces, or be, for two or three days, altogether suppressed. The calls to void it are more frequent than in health, especially at night and in the recumbent posture; the patient has to get up two or three times in the course of the night to empty the bladder. The urine is generally acid, surcharged with pigment; it often deposits the amorphous urates. Very rarely it is alkaline from fixed alkali. The natural urinous odour is lost; it has a faint unpleasant smell which has been compared to that of the washings of flesh.

The deposit when examined microscopically (see Fig. 40) is found to consist of blood-corpuscles, loose renal epithelium, free nuclei of these, tube-casts, shapeless masses of coagulated fibrine, and the broken *débris* of all these structures.

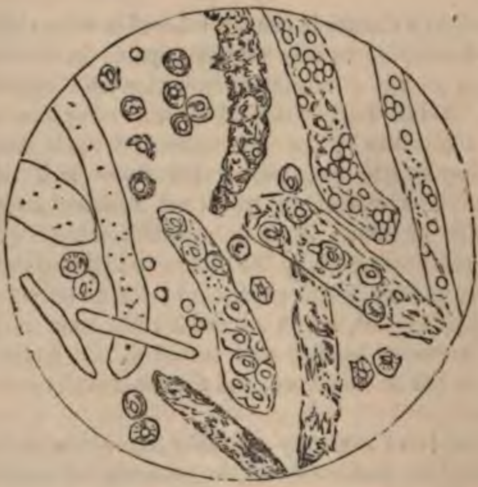

FIG. 40. Transparent, granular, blood- and epithelial casts from a case of acute Bright's disease; free renal epithelium; and blood disks.

There also generally found epithelial cells from the pelvis of the kidney and the bladder.

The renal epithelia vary a good deal in their appearance. Sometimes they look almost natural, only somewhat swollen and opaque. More frequently they are much broken down; their nuclei are set free, or are only invested in part by the granular cell-contents which naturally surround them. The disintegrated epithelium forms an amorphous dark granular *débris* scattered over the field. When very abundant, the epithelium communicates a milky appearance to the urine. The free nuclei greatly resemble red blood-disks both in shape and size, but they are devoid of the biconcave figure, and refract light more strongly. A solution of Magenta tints them of a deep carbuncle-red. The free blood-disks are frequently distorted. When the urine is of high density, they are shrunken, and often puckered at the margins ; on the other hand, when the urine is of lower density, 1017 and under, they expand, lose their central depressions, and eventually burst, and cease to be recognisable.

The tube-casts are abundant, and of varied size and appearance. The most common are of "medium" size, transparent, beset with epithelial cells or blood-disks. Mixed with these may be some "very large," and some "very small" hyaline casts, together with opaque granular casts (Fig. 40).

Specks of oil are generally altogether absent ; sometimes, however, a few small ones are seen either on the casts or within the epithelia; but their number is always quite insignificant in the early stages of the disease.

The proportion of albumen in the urine during the height of the complaint varies, according to Frerichs, from 8·2 to 12·7, 17·5 and 24·8 per 1000. Christison found 27 and Heller 57 per 1000. The quantity lost in the twenty-four hours varies from 80 to about 400 grains (Frerichs, Gorup v. Besanez). The natural solid constituents of the urine are diminished in proportion to the obstruction in the kidneys. The excretion of urea falls to 100 or 200 grains (from 400 or 500 grains in health) and the inorganic salts are considerably lessened. Uric acid maintains about its usual quantity.

The *blood* becomes speedily deteriorated by the unnatural drain through the kidneys. It becomes more watery and poorer in albumen, while urea, uric acid, and the extractives are unduly accumulated in it. The blood-corpuscles diminish in number as the disease proceeds. Fibrine is usually in excess, and the blood displays a buffy coat. The fat and inorganic salts retain their usual proportion. Frerichs supplies the three following analyses of the blood in the early period of acute Bright's disease :—

	I.	II.	III.
Specific gravity	1025	1022	1019
1000 parts of serum contained :—			
Water	908·10	915·88	938·9
Solids	91·90	84·12	61·1
Albumen	81·40	72·00	51·7
Fat	1·42	1·53	9·4
Extractive matters and salts	9·09	10·59	

After the disease has persisted for a variable period of a few days to some weeks, it proceeds to one of three terminations, viz., recovery, death, or lapse into the chronic state.

When the case is about to terminate favourably, the urine increases in quantity to three or four pints daily; its density falls below the natural mean (1012-1008); and the blood, renal elements, and albumen gradually diminish and finally disappear from it. At the same time, the skin becomes moist, and the serous effusions are re-absorbed. The rate of progress varies extremely. If albumen has totally left the urine in six weeks or two months, the recovery may be considered quick. The shortest period that I have known to elapse, from the first symptoms to complete re-establishment of the normal state, has been ten days.

Some cases reach final recovery only after a protracted and interrupted convalescence of many months. The urine during this period continues abundant, of low density, occasionally of pink colour from slight admixture of blood. The anasarca is also apt to recur and disappear, and recur again, perhaps several times, accompanied with febrile exacerbations of sub-acute character. In one such case observed by me the symptoms finally subsided in five months. The patient was seen ten months later, and the urine found perfectly free from albumen. In a second case, a slight admixture of blood continued, in diminishing quantity, for more than twelve months. In both these instances, and in a third similar to these, the characters of the urine were uniform: it was copious (three or four pints daily), of low density, slightly mixed with blood, slightly albuminous; the renal derivatives were devoid of fat, and, throughout the convalescence, comparatively scanty.

Not unfrequently, in the ordinary course of recovery from acute Bright's disease, the renal elements—both casts and epithelium—show slight signs of fatty changes. This circumstance is apt to embarrass the diagnosis, and lead to the suspicion of the existence of confirmed and chronic Bright's disease, if the case first come under observation in this stage. The doubt can only be solved by watching the progress of the case for a week or two.

But matters do not always take this favourable turn; and two new orders of symptoms arise, and bring life into imminent peril, or involve it in destruction. These are secondary inflammations of the serous membranes and the lungs, and uræmic intoxication.

Of the inflammatory complications, pericarditis is the most surely fatal, but it is rare. Pneumonia is more common; it breaks out without appreciable exciting cause, and usually runs a rapid course to a fatal end. Pleurisy and peritonitis are also not unfrequent, but greatly less to be feared. More or less bronchitis exists almost invariably. When the anasarca rises to an extreme degree, the integuments of the legs may inflame, and even mortify. These secondary inflammations are much more common in the later stages of chronic Bright's disease than in the acute disorder.

The uræmic phenomena are due to the retention in the blood of the excrementitious matters of the urine. They consist in a train of nervous symptoms—headache, vomiting, diarrhœa, convulsions, and coma—which are frequent incidents, and much to be feared in acute Bright's disease. They usually follow an excessive diminution or suppression of the urine from the increasing obstruction in the kidneys. It will be more convenient to postpone their consideration to a future section, when uræmia, in connection with Bright's disease generally, will be discussed.

Certain deviations from the usual course and symptoms are not unfrequently encountered. Although serous effusion generally first shows itself in the face, under the eyes, and then invades the trunk and extremities, it may begin elsewhere—in the feet, hands, or scrotum; or all parts of the body may swell up simultaneously. The effusion, too, may shift its place from time to time, or it may be poured out with disproportionate copiousness in certain localities (lung, pleura, submucous tissue of the glottis), and thereby determine sudden accession of alarming or fatal symptoms.

The anasarca commonly disappears some days or weeks before the blood and albumen have vanished from the urine; but sometimes the converse is the case, especially in individuals of lax frames and anæmic tendency. When cases of this latter class come under observation for the first time after the urine has become free from albumen, they are very apt to mislead, and their true nature can only be recognised by a careful sifting of the patient's previous history.

Diagnosis.—The general symptoms, and the alterations of the urine, are so significant during the height of the attack, that the disorder can scarcely be confounded with any other. But when the pyrectic stage is passed, and the case becomes protracted, there is often great difficulty in determining whether we have to deal with the declining periods of an acute and curable disorder, or with a disease which has already lapsed into the chronic and irremediable state, or with a disease which has been chronic from the first. Chronic Bright's disease is subject to occasional febrile recrudescences, which are deceptively like an attack of the acute disorder. The signs that the disease is acute and recent are:—free presence of blood and renal epithelium in the urine, absence of fat in the discharged elements, absence of long-standing complications, such as hypertrophy of the left ventricle, phthisis, caries, necrosis, and joint-disease. A careful consideration of the previous history and of the ostensible cause of the disorder is, also, of diagnostic importance. The less clearly a case can be traced to a definite exposure to cold, a bout of drinking, or to scarlet fever, or some other zymotic disease, the more reason is there, *pro tanto*, to fear that confirmed Bright's disease is established.

Prognosis.—Precise data concerning the fatality of acute Bright's disease are wanting. A large majority of the cases undoubtedly recover. Frerichs reckons the recoveries as two-thirds of the individuals attacked. Probably this proportion is below the truth if the scarlatinal cases be included.

The signs of approaching resolution are:—increased discharge of urine, diminished impregnation of it with blood and albumen, subsidence of the febrile phenomena, of the anasarca and serous effusions, and restoration of the cutaneous transpiration. At the same time, the countenance loses its stupid expression and its anæmic hue, and resumes its ordinary healthy aspect. The co-existence of all these signs leaves no doubt of advance toward a favourable issue; but the occurrence of some of them without the others, must not lead to too sanguine expectations. The anasarca may disappear totally, and blood cease to tinge the urine; the quantity of the secretion may increase considerably, the pyrexia pass away, and the general well-being of the patient improve greatly; but if the urine continue to contain a considerable amount of albumen, there is strong reason to apprehend that the disease is lapsing into a chronic state, or that the amendment is but a temporary lull in the symptoms, to be followed at no distant period by an exacerbation, which shall prove more disastrous than the original attack. Recovery cannot in any case be considered complete, until the urine has become perfectly free from every trace of albumen.

If the urine become progressively scantier, of higher density, and more abundantly charged with albumen, tube-casts, and renal epithelium, the worst consequences are to be feared. The advent of inflammatory complications, of œdema of the lungs or glottis, and, above all, of decided signs of uræmic poisoning, are of equally evil augury, and leave but slender hopes of the final preservation of life.

Etiology.—Acute Bright's disease, though not absolutely confined to any age, occurs, in the immense majority of cases, in childhood and youth. The individuals attacked are commonly of good previous health; in two instances, however, I have seen the disease complicated with acute pulmonary tuberculosis.

The exciting cause is usually some definite exposure to *cold* (a damp bed, wet clothes, lying or sleeping on the damp ground, sitting in a current of cold air, drinking cold water when in a state of perspiration), or a bout of drinking. A large proportion of the cases are sequelæ of scarlet fever, or (much more rarely) of some other zymotic disease. Some cases are due to pregnancy.

Treatment.—If the case is seen at the time of invasion, the patient should be at once confined to bed, swathed in flannels, and made to lie between the blankets. The loins should be immediately cupped to eight or twelve ounces (in children to two or three ounces). After the abstraction of blood, a large linseed-meal poultice should be applied, hot, to the loins, and changed every three hours. A hot-water bath or a hot-air bath should be administered every evening, or every second evening. When no conveniences for a hot-water bath exist, an excellent substitute is found in the "blanket-bath." A large thick blanket is wrung as dry as possible out of boiling water, and wrapped round the body of the patient; the bed-clothes are then heaped on. In twenty minutes or half an hour the hot blanket is removed, and the surface quickly dried with a warm soft towel.

The bowels should be freely acted on every other morning by an active purge, such as the compound jalap powder. An endeavour should also be made to allay the fever and restore the action of the skin, by citrate of potash draughts, given every two hours, in effervescence, or a mixture of the Liq. Ammon. Acet. in two or three drachm doses, with fifteen drops of tincture of henbane in an ounce of Inf. Lini. Dr. Barlow recommends tartar emetic in doses of from ¼ to ⅛ of a grain. I have myself employed the same remedy with the best effects, every four hours. Dr. Johnson also speaks highly of antimonial wine, sometimes combined with Dover's powder.

The diet should be composed of light farinaceous substances, with milk, beef-tea, and broths. Flesh meat in any form is objectionable in the early stage.

The abstraction of blood must be cautiously practised, on account of the tendency to anæmia in the later periods of the attack; and if the patient's health is broken by previous disease, or is constitutionally weak, even local depletion is better omitted. If severe headache, coma, or convulsions occur, the cupping may be repeated. In very threatening, sthenic cases, where the fever runs high, venesection may be practised.

When the fever has abated, and the anasarca is yielding, the more active measures should be discontinued, or pursued in a less active manner; but the efforts to restore and maintain the action of the skin should be persevered in. In the later periods, when convalescence has been fairly established, preparations of iron should be substituted for the alkaline and diaphoretic remedies. It is always well to begin with small doses, and to feel one's way. A too early resort to ferruginous preparations may be followed by a return of the acute symptoms. When iron is tolerated, it acts with great benefit, and hastens in a marked manner the disappearance of blood and albumen from the urine. My experience agrees with that of Dr. Parkes, that gallic acid exercises no beneficial influence in the acute disorder.

The use of mercury is objectionable, on account of the extreme susceptibility of patients suffering from Bright's disease, to the physiological effects of the drug. Severe salivation has been known to follow very small doses. In one of my patients two grains of blue pill, administered with extract of colocynth on two alternate mornings, produced profuse ptyalism.

The obstinate vomiting which occasionally prevails, may be combated with creasote, or small doses of chloroform, given in iced solutions. A careful revision of the diet should also be made. The gastric symptoms are sometimes due to direct sympathy with the renal irritation, and sometimes to genuine uræmic poisoning. The treatment of uræmia will be considered in a separate section.

The secondary thoracic inflammations present great difficulty in their management; they commonly set in when the patient is no longer in a fit state to bear the ordinary antiphlogistic means; and they run their course with unusual severity and rapidity. Counter-irritants and revulsives may, however, be energetically employed. Cantharides and turpentine should be avoided, from their special irritating effect on the kidneys; but hot-water applications, mustard poultices, and chloroform epithems may be applied locally over the chest, and to more distant parts—the calves of the legs, the feet, &c. Dry cupping over the chest is also a safe and sometimes valuable remedy.

When a favourable issue has been obtained, unusual care is required to guard against relapses, to which the patients continue liable for a considerable period. The slightest exposure is sometimes sufficient to reawaken the pyrexia, and to cause the reappearance of albumen and blood in the urine. A complete suit of flannels is essential; and as a rule, the convalescent should not be permitted to leave his room until the albumen has disappeared from the urine. When that comes to pass (or before, if the case prove very lingering), change of air to a warm sheltered locality is likely to be highly beneficial, and to hasten the restoration of the impoverished blood.

Objections have been made, on theoretical grounds, to the use of the saline diuretics (acetate and citrate of potash) in acute Bright's disease. Experience has proved, however, that they may be employed with great advantage. They become changed in the *primæ viæ* into alkaline carbonates, and these diminish the acidity of the urine, and render it more bland as it percolates the renal substance. In a disease which tends to spontaneous recovery under simple hygienic and prophylactic treatment, it is necessarily a matter of extreme difficulty to bring home the evidence of the curative power of drugs; but in a considerable number of cases of acute Bright's disease, coming under treatment early, I have obtained almost invariably the best results by the free administration of citrate of potash. And in no instance where the urine has been rendered alkaline in the first week of the complaint, have I observed the more severe uræmic symptoms, or secondary inflammations. In the later periods, when the fever has altogether subsided, while the urine still continues bloody and albuminous, the same medicament has not proved of any service in my hands.

Digitalis and broom-tops may be used freely in any stage to combat the dropsy. Dr. Christison recommends a combination of digitalis and bitartrate of potash as superior to either remedy given singly. "The former was usually given in the dose of one or two grains of the powder, in the form of a pill, three times a day, or in the dose of ten, fifteen, or twenty minims of the tincture, three times daily in a little distilled water of cinnamon or cassia. The cream of tartar was administered thrice a day in the quantity of a drachm and a half, or two drachms, with about five ounces of water. Diuresis may generally be induced by such means in the course of three or four days, sometimes sooner—seldom, however, if delayed beyond the seventh day." (p. 149.)

Hamburger speaks strongly in favour of quinine in scarlatinal dropsy, after the pyrexia has abated. He gives to children 1½ or 2 grains, and to adults 3 to 4 grains, twice a day. Of 47 severe cases thus treated he obtained amendment in 44, either immediately or in a few days (Prag. Vierteljahrsch. 1861).

CHAPTER IV.

CHRONIC BRIGHT'S DISEASE.

(*Chronic Diffuse Nephritis.*)

See references prefixed to Chap. II., p. 293.

ANATOMICAL CHANGES IN THE KIDNEYS.

THE kidneys of persons dying of chronic Bright's disease, present three chief types of alteration, viz. :—

TYPE I.—*Kidney smooth, white,* and enlarged ; in extreme cases, rarely met with, kidney atrophic.

TYPE II.—*Kidney granular, brownish or red,* and contracted.

TYPE III.—*Kidney lardaceous or waxy* (so-called *amyloid* degeneration).

The special clinical history pertaining to each of these anatomical types, has not been made out with sufficient precision to enable them to be invariably recognised during life ; but much light has, in recent years, been thrown on the subject, enough to permit a synopsis of the symptoms, and conditions of origin, of the three types to be presented.

1. *Smooth white kidney.*

The structural changes in the smooth white kidney, are similar to those already described as pertaining to acute Bright's disease, but advanced to a further stage ; the surface continues perfectly smooth ; the organ is greatly enlarged ; and the capsule is thin and easily stripped off. If, however, the patient survive sufficiently long—which very rarely happens —the enlargement gives place to a progressive dwindling ; and in very extreme cases, the kidney may be thus reduced to a weight of only an ounce, or even less. This dwindling is not, it seems, an invariable event, even when the patient survives for some years. Dr. Wilks relates the following example :—

"A young woman, æt. 23, had scarlatina *three* years before death. There was very slight eruption; dropsy soon followed, which lasted a year. Then the patient was slightly better, but remained an invalid, with œdema of the legs, until the last five months, when very extensive and general dropsy came on and persisted. The urine was then scanty, dark, and contained exudative deposit. She had three epileptiform fits, and death subsequently ensued from pleuritis and pericarditis. Lungs were found very œdematous. The aorta and arteries covered with an atheromatous deposit: and the *kidneys* were large and white, with an abundance of deposit, much of which had undergone a fatty change." (Guy's Hosp. Rep. 2nd series, vol. viii. p. 243.)

When the smooth kidney becomes atrophied, the capsule is slightly thickened and disposed to adhere to the renal surface.

The progress of the disease in the interior of the gland is thus described by Dr. Dickinson :—

"The overstretched tubes, one after another, burst, and their contents, no longer secluded by a surrounding membrane, are brought within the ready operation of the absorbents and removed. This however is a matter of inference rather than of demonstration. Examining in section a number of enlarged smooth kidneys, I found that about half of them had thus commenced to pass from the stage of enlargement to that of decrease, although there was nothing in their outward appearance to indicate it, their bulk being still excessive." In a section taken from such a kidney, are found "places, especially near the surface, where the Malpighian bodies, which have now become enlarged and enveloped by condensed fibrous tissue, lie in unnatural proximity. Often several almost touch each other. Their intervals are filled up by the *débris* of collapsed and ruined structure. Shrivelled remnants of the tube-membrane are generally clearly seen. This condition is as yet only partial. A distended duct is often seen winding among the wasted remains of its companions. The tubes, one after another, collapse until a great portion of the once enlarged gland has been reduced to the bulk of little more than its Malpighian bodies. The appearance of the organ affected has by this time undergone some alteration. It has lost its excess of bulk, and even shrunk below the natural size. . . . When the organ is cut open, it is manifest that it is chiefly composed of cone substance: an edge, perhaps, no thicker than a shilling, being all that separates that structure from the capsule. What remains of the cortical material is coarse in texture, pale, and firm." This condition—that of the smooth dwindled kidney—is rarely attained, because the patient seldom survives sufficiently long. Of 2350 bodies examined in St. George's Hospital in ten years, it was only found in twelve instances.

The epithelium in the smooth kidney is found disintegrated, atrophied, and very commonly in a state of fatty degeneration. When this change acquires a high degree, the condition denominated "fatty kidney" is attained. The cones undergo changes of a corresponding character, but less developed; and fibrinous casts are found occupying the interior of the tubes.

Synopsis of symptoms and conditions of origin.—The smooth kidney here described, may be expected in those cases in which chronic Bright's disease has followed on the acute disorder. The invasion of the disease has been sudden, and it can usually be traced to some definite exciting cause. I have also seen the large white kidney in chronic Bright's disease following repeated pregnancies, and in a case arising in the course of phthisis.

The average age of 106 cases of smooth large kidney, examined by Dickinson, was 28·2 years; in 11 cases of smooth dwindled kidney the average age was 43·6 years; whereas in 250 cases of granular kidney the average age was 50·2 years.

Serous effusion is an almost invariable coincidence; the body is commonly bloated with dropsy; the face pale and puffy, and the cutaneous surface conspicuously white, smooth and glossy. There is also a markedly greater tendency to secondary inflammations, and to uræmic accidents, than in granular kidney; but less to valvular heart disease and hypertrophy of the left ventricle.

The urine is commonly bloody from time to time, highly albuminous, scanty in amount, and of a specific gravity not departing much from the standard of health. Epithelial elements and tube-casts are usually abundant in the urine, and often fatty. Cells having the appearance of pus corpuscles, are common toward the later periods.

The disease is of shorter duration than granular kidney. In fatal cases, the ordinary duration of the disease is under six months. Out of 34 cases observed by Dickinson, 26 died under six months, and only two survived the year. Occasionally, temporary (apparent) recoveries and succeeding relapses protract the disease for a longer period; and, quite exceptionally, the disease may last for years. Dr. Johnson records an instance which endured for ten years, with good preservation of health for a portion of that period. Nine years before death, the urinary deposit clearly indicated fatty degeneration of the kidneys. After death, the kidneys were found dwindled to an ounce and three-quarters for the pair.*

2. *Granular red kidney.*

The gland is diminished in size, and reduced to a weight of two or three ounces, or less. Its surface is rough, and beset with numerous rounded elevations, varying from the size of a pin's head, to a hemp-seed, or even a small pea. The capsule is opaque, thickened, and adherent to the subjacent surface, so that it cannot be peeled off without tearing the glandular structure. In certain spots the capsule sinks into the substance of the cortex, and divides the kidney irregularly—giving it a lobular appearance. On section, the cortex is manifestly atrophied, as compared with the cones, and forms a thin rim of only a line, or less, in thickness around the bases. It has a red, or brownish-red colour, and a coarse granular texture. The entire organ is tough and resistent.

It is only rarely that the granular kidney is encountered in the early stage of its development. When such is the case, thickening of the capsule and slight granulation of the surface are found to precede contraction, so that the organ at this period preserves its normal volume. The granular kidney, in the contracted state, allows injections to penetrate imperfectly. Dickinson found that when a stream of warm water was propelled through the blood vessels, a very considerably less quantity passed in a given time than through a healthy kidney—

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* Med. Chir. Trans., vol. xlii., p. 160.

less also than through the large smooth kidney—showing that the permeability of the gland to the blood-stream was greatly lessened. In the healthy kidney, the mean discharge through the renal veins in ten minutes was 119 ounces; in the large smooth kidney, 90 ounces; and in the granular contracted kidney, 25 ounces.

When a thin section of a granular kidney is placed under the microscope, the secreting tissue is found to have undergone extensive destruction. The Malpighian bodies are shrunk to half their size, and unnaturally crowded together. Their vascular tufts are embraced in a fibrous and granular investment, and, in extreme instances, compressed into an impermeable knot at the bottom of their capsules. The uriniferous tubes are altered in various ways, and to various degrees. Some are denuded of epithelium and reduced to mere tubular threads; others, equally denuded, contain glassy fibrinous cylinders; while others are crammed with broken-up epithelium. Oil is found not unfrequently both in the fibrinous exudation and in the disintegrated epithelium, but not so commonly nor so abundantly as in the smooth kidney. Amid tubes changed to this degree, are found others not much altered, and lined with their proper and healthy epithelial investment. Between the wasted structures lies a large quantity of adventitious connective tissue, which gives the organ its peculiar toughness.

Of the intimate nature of the process which finally brings about this result, there are conflicting opinions. Dr. Johnson believes that the mischief begins in the epithelial cells, while Dickinson describes it as originating in the intertubular structure, and as consisting in an effusion, which afterwards becomes converted into a fibrous material. According to the last-named observer, the effusion is first thrown out beneath the capsule, and then penetrates at certain spots, as fibrinous processes, into the interior of the cortex, athwart the convoluted tubes, which it involves in destruction. The points where the fibrinous processes penetrate are depressed, and when these are numerous and distributed with tolerable regularity, the result is superficial granulation. The disease thus travels from the surface towards the central parts, and eventually involves the pyramids.

The development of cysts is much more common in the granular than in the smooth kidney. They vary in size from a pin's-head to a pea or a hazel-nut; but many are so minute that they can only be detected by the microscope, not being larger than the width of the uriniferous tubes. Mr. Simon believes that they are formed by an immense dilatation of epithelial cells—a development that seems incredible, considering the fragility of the outer portions of these cells; and one that would be, so far as I know, without parallel in histogenesis. A better sustained, and more commonly accepted, view is, that they are produced by obstruction of the uriniferous tubes with exudation, at intervals, or by compression of their walls at interrupted spots by the contracting adventitious tissue. The spaces thus enclosed become distended with a serous fluid, and are sometimes found lined with an epithelial layer. Their contents are not urinous, but consist of an albuminous saline solution. In the cones they are sometimes elongated and placed end to end like a string of sausages (Dickinson). (See Cysts and Cystic Degeneration in the Kidneys.)

Synopsis of symptoms and conditions of origin. The granular kidney is found in the vast majority of those cases of Bright's disease, which aer chronic from the beginning—those which commence insidiously, without definite exciting cause. Dropsy is altogether absent in a large proportion (perhaps in a quarter —according to some, in one-half—) of the cases, and when present, it is commonly slight, and limited to œdema of the ankles and legs, or a puffiness under the eyes. It often disappears for a while, and returns again.

The disease may run a latent course for months and years. A deep constitutional cachexy is associated with it in a large proportion of cases. The subjects of it are more advanced in years than those of the smooth large kidney (see p. 318). The cutaneous surface, though pale and anæmic, has not the conspicuous whiteness of the preceding type, and the features are often pinched and sallow. Cardiac hypertrophy is a very frequent concomitant.

The urine is copious—three or four pints a day—and of low specific gravity; the quantity of albumen is comparatively slight; in rare cases it may even be temporarily absent from the urine. Toward the termination of the disease, however, the urine becomes scanty, or even suppressed. The deposit is slight, composed of hyaline and granular casts, with very slight admixture of epithelium, not often fatty: the deposit is often so scanty that it may escape detection, or there may really be none. As a rule, blood is absent.

The common predisposing causes are habitual intemperance, gout, and extensively distributed fatty degeneration of the tissues.

As these two types—the smooth large white kidney, and the granular red contracted kidney—constitute the vast majority of cases of Bright's disease, the question of the oneness or multiformity of Bright's disease has mostly been limited to the inquiry—whether the latter is the ultimate stage of the former, or whether the two are distinct from first to last.

Reinhardt and Frerichs believe that the large white kidney will, if the patient survive, eventually become granular red and contracted. This view seems latterly to have gained common assent in Germany, and in Rosenstein's recent work it is adopted without discussion. In this country, however, the opposite view has steadily gained ground; and the evidence brought forward by Johnson, Wilks, and Dickinson appears to place the matter beyond reasonable doubt. Dickinson contributes the table on the following page, which shows the great difference which exists between the relative frequency of certain secondary affections in the two types.

Johnson found that out of 26 fatal cases of enlarged kidney, observed by himself, there was dropsy in 24, or 92 per cent.; whereas in 33 fatal cases of contracted kidney, there had been dropsy only in 14, or 42 per cent. He pertinently observes:—"If all the contracted Bright's kidneys have passed through a previous stage of enlargement, it is difficult to understand how it can happen that the majority of those patients who have reached the final stage of renal degeneration should escape the dropsy, which, in a greater or less degree, troubles nearly all those who die in what is assumed to be an earlier stage of the same disease."*

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* Med. Chir. Trans., vol. xlii. p. 156.

TABLE *showing the relative frequency of the various secondary affections in the two main types of Bright's disease—after Dickinson.*

	Bronchitis.	Diarrhœa.	Vomiting.	Pleurisy.	Pericarditis.	Peritonitis.	Pneumonia.	Œdema.	Ascites.	Hydrothorax.	Hydropericardium.	Epistaxis.	Erysipelas.	Cirrhosis of liver.	Atheroma.	Hypertrophy of heart.	Valvular disease.	Convulsions.	Simple coma.	Sanguineous apoplexy.
Smooth kidney, 119 cases . . .	18·5	20·1	26·0	64·3	27·7	25·2	20·1	66·3	33·6	28·5	24·3	0·8	15·9	10·9	21·8	24·3	17·4	10·9	10·0	2·5
Granular kidney, 250 cases . . .	22·0	11·6	15·6	60·8	32·8	13·6	12·4	48·0	26·0	25·2	14·0	0·8	6·8	15·2	52·4	48·0	43·2	5·6	8·8	6·8

Œdema, peritonitis, pneumonia, and convulsions are seen to be much more frequent with the smooth kidney; whereas heart disease, atheroma, and apoplexy are much more frequent with the granular kidney.

It must, of course, be borne in mind that it is not now disputed that the large white kidney does sometimes suffer atrophic changes, and that in a few exceptional cases it may at length dwindle to very small dimensions. Both Johnson and Dickinson adduce several examples of such a change, but they insist, that even in its furthest stage of contraction, the smooth white kidney is still distinguishable from the granular red kidney.

3. *Lardaceous or Waxy Kidney.*

(So-called Amyloid Degeneration of Virchow.)

Most pathologists now distinguish the waxy or lardaceous kidney from the preceding varieties of Bright's kidney.

Externally, the waxy kidney is smooth, or sometimes slightly roughened; the capsule peels off readily. The organ is usually enlarged, sometimes, however, diminished in size. On section the appearance is characteristic. The cortex is bloodless, of a white or yellowish colour, with a waxy, smooth, translucent appearance, resembling bacon-rind. The organ is conspicuously tough and hard. On the smooth cut surface, little appearance of the natural secreting structure is seen, but it is dotted over with bright glancing points: these are the changed Malpighian bodies. The cones appear unnaturally red and distinct.

This description answers only to extreme degrees: in slighter cases the nature of the change can only be clearly made out with the microscope. When a thin section is highly magnified, the waxy material is seen to affect mainly (sometimes exclusively) the blood-vessels. The deposit invades primarily the muscular coat of the arteries, which it renders abnormally thick and transparent, thereby diminishing the lumen of the vessel. The Malpighian corpuscles are the parts earliest attacked. They appear as shining particles with thickened capsules; their vascular tufts are infiltrated with the waxy material. In advanced cases, the vasa afferentia, with the arteries and capillary network of the cortex, and even the vessels of the pyramids, are similarly changed.

The epithelial cells of the uriniferous tubes are commonly withered, often infiltrated with fatty molecules; but they are never themselves, according to Wagner, the seat of waxy change.* Hyaline waxy casts exist in some of the tubules. The stroma of the gland is much increased in quantity, but is not lardaceous in character.

Such a kidney gives the peculiar reaction of the so-called amyloid substance of Virchow—it assumes a deep red with watery solution of iodine ; and this tint is changed to violet on the addition of sulphuric acid.

The liver and spleen are usually greatly enlarged, and in a lardaceous state, when the kidneys are so affected. Of 77 cases collected by Rosenstein, the three organs together were affected in 48 ; the spleen and kidneys in 20 ; the liver and kidneys in 4 ; and the kidneys alone in 5 cases.

The chemical nature of the waxy material has only recently been investigated. Virchow concluded from the violet colour produced by iodine and sulphuric acid that it belonged to the same group as starch and cellulose, which likewise yield a violet tint with the same re-agents. But the ultimate analyses of C. Schmidt and Kekulé show that it contains nitrogen—and indeed as much as 15 per cent., or almost exactly the same proportion as the protein compounds. Neither of the chemists named could produce a particle of sugar from it by boiling with dilute sulphuric acid. It further resembled albuminous compounds, in yielding a violet colour with the cupro-potassic solution, in dissolving completely in dilute caustic potash, and in being precipitated from this solution in white flocks by acids.† The proportion per cent. of carbon, hydrogen, and nitrogen, found by Kekulé, in purified waxy matter, from an exquisite specimen of lardaceous spleen, was :—C 53·58 ; H 7·00 ; N 15·4—which corresponds closely with the per-centage of the same elements in albumen. The correspondence is so close that it does not even permit the supposition, that, like Chitin, lardaceous matter may consist of a combination of a hydrocarbon with an albuminoid substance. To call it "amyloid" is simply a misnomer, and an unfortunate one, because it leads to confused notions as to the existence of some connection between waxy degeneration, and the (genuine) amyloid substance found in the liver.

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* Dr. G. Stewart states that, occasionally, epithelial cells are found on the casts in the urinary deposit, which exhibit the peculiar reaction of lardaceous matter.

† Friedreich says that the "amyloid reaction" (with iodine and sulphuric acid) was obtained by him in perfection, with decolorized fibrine from an old hæmatocele.

Synopsis of symptoms and conditions of origin. Lardaceous degeneration of the kidneys always comes on insidiously, and in cachectic persons, debilitated by some pre-existing wasting disease. In 100 instances collected by Rosenstein it co-existed with the following disorders :—

Pulmonary tubercle (combined, in 10 cases, with caries of bone, and in one case with syphilis).	44	cases.
Caries	29	,,
Syphilis	15	,,
Emphysema	3	,,
Cancer	3	,,
Psoas abscess	2	,,
Pyelitis and hydronephrosis	2	,,
Abscess of liver	1	,,
Chronic alcoholism	1	,,

The aspect of patients with waxy kidneys is pale and cachectic, and the course of the disease is essentially chronic. Dropsy is present in the majority of the cases (in 61 out of 72 collected by Rosenstein); in some it is abundant and general, in others slight and partial. Uræmic symptoms are strikingly infrequent.

The urine, in the early stage, is abundant and only slightly albuminous, but toward the close, it becomes highly albuminous, scanty, and of high density. In a case under my observation, the quantity of albumen was remarkable for its variability from day to day.* The urine is commonly pale, and allows only a very scanty deposit to subside. This consists of atrophied renal cells, which are sometimes fatty. Cells resembling those of pus are occasionally found, either separate or aggregated round a cast. The tube-casts are usually waxy and hyaline; they do not, according to Dr. G. Stewart, yield a violet colour with iodine and sulphuric acid. Epithelial casts are not unknown in these cases. Münch detected "corpora amylacea" in the urine of a man with lardaceous kidney ; they were constantly present, and were coloured violet by iodine and sulphuric acid.*

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* A case is reported by Pleischl and Klob, in which the urine was throughout free from albumen—cited by Rosenstein.

The diagnosis of waxy kidney rests chiefly on the co-existence of enlarged (lardaceous) liver and spleen, and on the presence of one of the wasting diseases, of which waxy kidney is known to be a frequent complication, namely, phthisis, cariês, long-continued suppuration, and constitutional syphilis. The characters of the casts are not diagnostic, although they may be suggestive. Dr. Stewart lays considerable stress on the abundance of the urine in the early period, and on a leukæmic state of the blood. Rosenstein denies that the urine is invariably abundant in the early stage.

GENERAL COURSE AND SYMPTOMS.

Chronic Bright's disease, in the great majority of instances, begins slowly, imperceptibly. It is rarely detected until it has already existed some months—it may be, years. The attention of the patient is at length awakened by the gradual failure of his strength, the increasing pallor or sallowness of his complexion, and his disinclination to exertion; perhaps his suspicions are aroused by a little puffiness under the eyes, a slight swelling of the ankles at night, unusually frequent calls to void urine, or shortness of breath.

In other cases, these premonitions are altogether wanting, or perhaps they pass unheeded. The fatal disorganisation in the kidneys proceeds silently, amid apparent health, and then, suddenly, declares itself by a fit of convulsions, rapid coma, amaurosis, pulmonary œdema, or a violent inflammation.

Or, again, the disease creeps on stealthily in the wake of some pre-existing chronic disorder—phthisis, caries, necrosis, joint-disease, constitutional syphilis, chronic alcoholism, or exhausting suppuration.

Or, it may be a continuation or sequela of acute Bright's disease.

Lastly, the disease may lie concealed for an undetermined period, and then reveal itself after exposure to cold or a fit of intoxication, in the guise of an acute attack—with rapid general anasarca and scanty sanguineous urine.

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* Cited by Parkes. *Composition of the Urine*, p. 394.

The principal symptoms of the disease are:—albuminous urine; deposits of tube-casts and renal epithelium; dryness of the skin; frequent micturition, especially at night; dropsical effusions into the subcutaneous cellular tissue, serous cavities, or pulmonary substance; derangements of digestion; progressive hydræmia; uræmic phenomena (headache, amblyopia, convulsions, coma, vomiting and diarrhœa); hypertrophy of the left ventricle; secondary inflammation of the parenchymatous organs and serous membranes.

Few cases present the whole of these symptoms; and many present only two or three of them. The alterations in the composition of the urine are the most invariable; they are also the earliest and most distinctive symptoms; next follow, in the order of constancy, the deterioration of the blood, the dropsical symptoms, and lastly the uræmic and inflammatory incidents.

The disease usually pursues an interrupted course. It is subject to exacerbations from time to time, with intervals of quiescence. The exacerbations are generally occasioned by exposure to cold, or some imprudence in diet or regimen: sometimes no cause can be assigned for their occurrence. They are marked by pyrexia; and resemble, often closely, an attack of acute Bright's disease. The intervals of quiescence may be longer or shorter, some weeks or months, or a few years; the remission of the symptoms is commonly only partial—the main features of the disease persisting, though in diminished prominence. Sometimes, however, the remission is almost complete, and there remains little except the albuminous state of the urine to attest the existence of renal mischief. Nay, even this may, in very exceptional cases, be absent, and the nature of the case be first revealed at the autopsy.

After each exacerbation, it is commonly pretty evident, that the disease has taken a step in advance, and assumed a fuller development; and that, probably, an additional portion of the kidney, hitherto spared or only slightly affected, has been disabled.

But whether it thus proceed *per saltum* or more continuously, the kidneys are at length so deeply injured, and their depurative functions so far abrogated, that life falls a forfeit.

The immediate cause of dissolution is various. Sometimes the sufferer passes peaceably away exhausted by anæmia, burdensome anasarca, and defective digestion of food. More frequently the final scene is tumultuous. Two of the cases to be hereafter related terminated, amid a pyrexial exacerbation, with formation of clots in the heart. About one-third of the subjects of chronic Bright's disease perish by uræmic poisoning, either in the form of coma and convulsions or irrepressible vomiting and diarrhœa. A considerable number die from the dangerous situation, or intensity, of the dropsical effusion—as when the lungs or glottis are invaded; or death comes from hydrothorax, or from gangrenous erysipelas set up in the tense hydropic integuments of the thighs, legs, or genitals. About one-fifth die by secondary pneumonia, pericarditis, or double pleurisy. The remainder are cut off by less closely connected complications—apoplexy, cirrhosis, phthisis, intestinal ulcerations, etc.

From the difficulty of assigning the exact date of invasion, the *duration* of the disease can only be approximately ascertained. Enough is, however, known, to show that it varies within very wide limits. The usual period is from two to three years; but cases may end in six months, or be protracted for four and five years. Exceptional instances have been recorded, in which patients have survived 10 years (Johnson and Kussmaul), and even 15 (Bright) and 23 years (Oppolzer).

The following abstracts of cases will serve to exhibit the broad features of the disease, in its more familiar aspects; and prepare the way to a more detailed consideration of the symptoms and complications:—

CASE I.—*Chronic Bright's disease, latent two years, without dropsy—fatty casts and cells in the urine. Death by uræmic convulsions.*

Mr. V., a solicitor of temperate habits, æt. 50. Two and a half years ago, Mr. V. suffered from sciatica, for which he was under medical treatment. At that time a little albumen was discovered in the urine, but slight importance was attached thereto. Mr. V. speedily recovered from his sciatica, and continued in good health, attending to his business, until four months ago, when he became subject to shortness of breath and catarrhal symptoms. These were not severe enough to prevent the patient from pursuing his occupation, until the beginning of April, 1864, when I was requested to see the case with Mr. Mellor. The symptoms complained of, were, shortness of breath on exertion, and failure of strength; there was not a particle of œdema (nor had there ever been any) nor ascites. The liver and spleen were not enlarged; there were no cardiac murmurs; but there existed slight præcordial bulging, and the heart's apex beat in the vertical line of the nipple. The shortness of breath evidently depended on pulmonary œdema. The countenance was pale and sallow, and the body spare, but not conspicuously emaciated. The urine was copious (three pints), of low density (1012), and highly albuminous ($\frac{1}{3}$); it deposited a not inconsiderable flour-like sediment, composed of casts and renal epithelia, many of which showed abundant signs of fatty charges (see Fig. 41):—

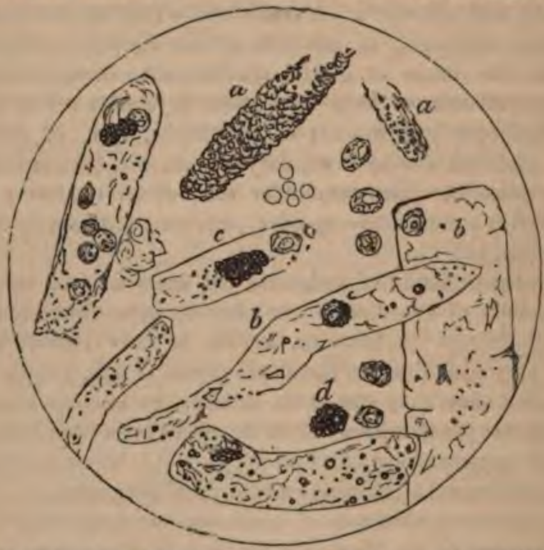

FIG. 41. Casts and renal cells from the urine of Mr. V——. *a a.* granular opaque casts; *b b.* hyaline casts; *c d.* fatty masses.

The casts were mostly medium-sized: some were granular and opaque, as at (*a*); others, in about equal numbers, were nearly hyaline, with only very faint markings, as at (*b*). Withered epithelia studded some of the casts, or lay scattered free about the field. Botryoidal fat-masses lay embedded in some of the casts; other casts were dotted over irregularly with oily particles. Some of the renal cells were similarly dotted in their interiors, while others were entirely changed into round agglomerations of fat molecules (granular corpuscles) (*c*, *d*). A few sparse blood-disks were scattered about.

The previous history was singularly barren of etiological indications. The patient's mode of life had been strictly temperate; and there was no evidence of repeated exposure to cold, nor of gout. Father and mother died at the age of forty-five—the latter of consumption. He himself had enjoyed remarkably good health, until the invasion of his present complaint.

The treatment adopted was:—dry cupping the chest, warm bath every second day, flannel clothing, cod-liver oil and iron. The dry cupping removed the dyspnœa at once, and some general amendment took place in the course of the ensuing month.

This gentleman continued under observation until his death, which took place in three months. He improved for a while, and was able to go to Southport for a fortnight, where he derived considerable benefit. He considered himself so well on his return, that he believed a week or two would complete his recovery. He resumed his usual occupation, and, for a week or two, went daily to his office. But this truce was wholly deceptive; the condition of the urine never improved. It became progressively scantier in quantity—first it fell to 40 ounces, and then to 30 ounces, while the specific gravity continued to range from 1009 to 1011; and the deposit of casts became more and more opaque-granular, and less and less fatty. Emaciation also progressed, and the shortness of breath returned, and could no longer be kept under by dry cupping. A persistent feverishness began to prevail: the nights were restless; but during the day the patient was dull, almost drowsy, and indifferent. Not a trace of œdema appeared throughout the complaint. The hypertrophy of the heart became progressively more conspicuous.

In the last fortnight of life, the urine became very scanty (still of low density), and was totally suppressed for twenty-four hours before death; vomiting recurred frequently, with utter loathing of food, and especially of animal flesh. The sight failed, and two days before death he became completely blind for more than half an hour. The restlessness increased, accompanied with wandering delirium, the tongue became dry, the indifference merged into drowsiness, and, after a fit of convulsions, he died.

The general course and symptoms clearly indicated a granular contracting kidney: but the friends would not permit a *post mortem* examination.

Case II.—*Chronic Bright's disease from intemperate habits—sudden anasarca after a wetting. Death from pericarditis. Granular contracted kidneys.*

W. M., a carter, æt. 40, of intemperate habits, was admitted into the Royal Infirmary, March 1, 1858, with general anasarca and ascites. He had followed his employment, and considered himself in good health, until three months back, when he got a severe wetting, and allowed his clothes to dry on him. Soon after followed lumbar pains and general swelling of the body. On admission there was œdema of the face, trunk, and extremities, and considerable ascites. The skin was dry; the urine, of low specific gravity, contained tube-casts, but no blood. After he had been in the house a fortnight, the urine became scanty, and intense pericarditis set in, which proved fatal on the fifth day. He died comatose with suppression of urine. At the autopsy the kidneys were found granular and greatly atrophied; scarcely any cortical substance remained. Abundance of fibrinous exudation existed in the pericardium; left ventricle immensely hypertrophied—the walls fully one inch thick; the valves were healthy.

The state of the organs after death, indicated that the disease had been really in existence for a much longer period than the few months during which symptoms had been noted by the patient.

CASE III.—*Chronic Bright's disease from repeated pregnancies—recurrent uræmic convulsions. Granular contracted kidneys.*

Mrs. X., æt. 39, became pregnant of her sixth child in the autumn of 1862. About the third month, unusual frequency of micturition at night was observed, and soon after slight œdema of the face and legs. The urine was found to contain albumen. The fœtus was expelled without accident at the fifth month, and a few days after all the œdema disappeared; but the urine still continued albuminous. I first saw her about two months after the miscarriage. There was no œdema of any part. The urine was of low density, and moderately albuminous. The deposit contained a few transparent tube-casts, some of which showed slight evidences of fatty change; others were opaque, and studded with withered epithelia (see Fig. 42):—

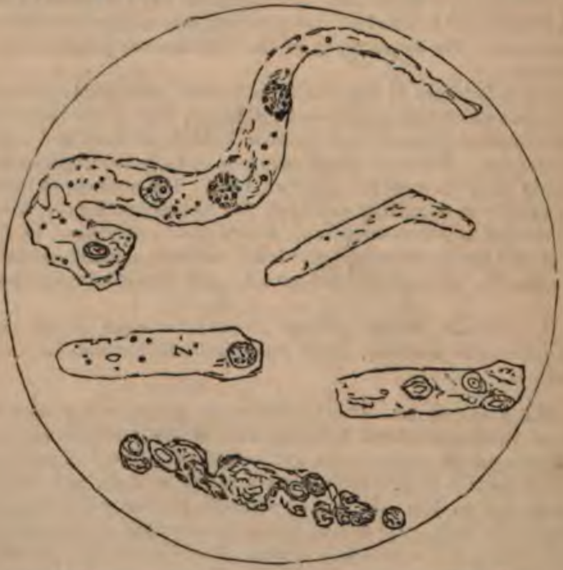

FIG. 42. Transparent and opaque casts from the urine of Mrs. X——.

The patient continued under observation for above a year, and died, at length, comatose, after repeated attacks of convulsions. Each catamenial period was marked by great nervous excitement; and on several occasions convulsions took place at these periods, accompanied with temporary amaurosis. Severe headache was a very constant symptom, especially on the days preceding the catamenial periods. After death the kidneys were found granular and atrophied, and the left ventricle much enlarged.

CASE IV.—*Chronic Bright's disease from intemperance and exposure to cold—general dropsy, complicated with old chronic peritonitis. Death from syncope. Smooth white kidney, beginning to contract. Myriads of minute uric acid calculi in the infundibula.*

J. R., æt. 48, a French polisher, from Oldham, was admitted into the Royal Infirmary, April 4th, 1864.

There was great ascites, tense œdema of the lower extremities, with an erysipelatous state of the integuments of the upper and inner parts of the thigh and scrotum; œdema also of arms and back of hands. The heart was displaced upwards, and much enlarged; there were no cardiac murmurs. There was great emaciation, cough, purulent expectoration, and orthopnœa.

The urine was scanty, dark-coloured from blood, highly albuminous; it let fall an abundant chocolate-coloured deposit, composed of "blood-casts," "granular casts," and "epithelial casts," with abundance of free renal epithelium and free blood-disks. Mixed with these were a large number of irregularly-tailed and spindle-shaped cells, evidently from the pelvis of the kidney (see Fig. 43.)

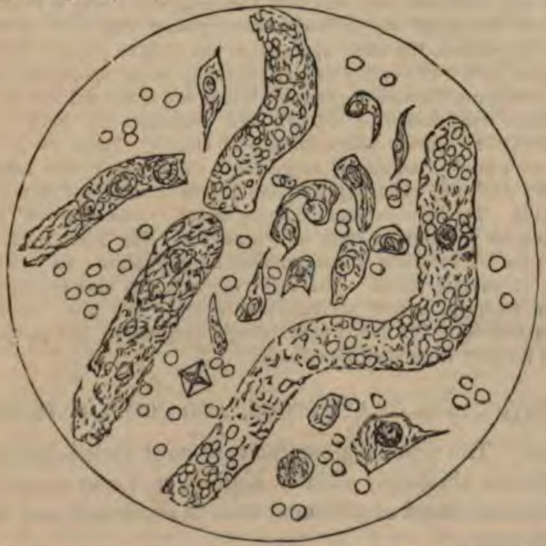

FIG. 43. Blood-casts, granular casts, blood-disks, tailed and irregular cells from the pelvis of the kidney—from the urine of J. R.

The patient stated that he had been ailing twenty weeks: the symptoms had come on gradually. The swelling had first appeared in the belly, and the enlargement of the abdomen was still out of proportion to the general dropsy. His habits had been for years intemperate, and he was often exposed to chills, in suddenly passing from his warm workshop to the cold open air. He had, however, been a healthy man, and had never lost a day's work until his present illness.

He went on, with little change in the general symptoms and urine, for twenty-five days, when he became feverish and delirious, apparently from cold, taken by imprudently exposing himself after a warm bath. He suddenly fell back dead on April 27th, as the nurse was shifting him for the purpose of making his bed.

Autopsy, 24 hours after death. There was a good deal of anasarca of the lower limbs, fore-arms, and hands. An enormous quantity of serum escaped from the peritoneal cavity. The peritoneal membrane, in its entire extent, was thickened, or rather, it was invested with a layer of thin adherent fragile false-membrane of a pearly translucency, like the hyaline membrane of a hydatid sac. The intestines were sunk on the spine; there was no recent peritonitis. The liver was covered over with a rough layer of hyaline false-membrane, which evidently embraced it tightly, and had caused it to shrink much below its natural bulk. On section it did not display a cirrhotic structure. The spleen was rather large; its capsule thick and opaque. The kidneys weighed together eleven ounces. They were firm, and their capsule smooth, but opaque and thickened. The capsule peeled off with only moderate ease, and tore the subjacent tissue a little. The surface of the gland was yellowish-white picked with dead white, like ivory. On section, the same appearance was seen to prevail throughout the cortical part. The cortex was, if anything, below its normal proportion. The pyramids were of a faint red colour, not unnatural-looking. The infundibula were somewhat dilated, and contained (in both kidneys) myriads of very minute yellow, uric acid calculi. These varied in size from a pin's-head to an almost microscopic object; they were lumpy and irregular in shape. The papillæ were flattened, some of them almost obliterated. The kidneys were evidently of the "smooth white" species, beginning to pass into a state of contraction. The heart weighed 12½ ounces; the left ventricle was enormously hypertrophied; its walls seven-eighths of an inch thick. The right ventricle was also hypertrophied, and the tricuspid orifice somewhat patulous. All the valves were perfectly healthy. The lungs were strongly compressed, and partially airless and leathery from pleuritic effusion.

PARTICULARS OF SYMPTOMS, AND COMPLICATIONS.

Urine. The urine is albuminous to most varied degrees. It may become absolutely solid on boiling, or it may contain only the minutest traces of albumen, even in confirmed and fatally-tending cases.

Absolute freedom from albumen, even for short intervals, is very rare: I am convinced, that a considerable number of the cases so reported, are examples of imperfect testing. When the quantity of albumen is extremely small, nice management is required to detect it. Heat is insufficient; nitric acid should be allowed to trickle slowly to the bottom of the test-tube, and some *minutes* allowed to elapse in order to develop the hazy zone above the level of the acid. But it must be admitted that chronic degeneration of the kidneys, not distinguishable from some forms of Bright's disease, *do* exist under certain circumstances, without albuminuria. The following example of scarlatinal dropsy, running a chronic course and ending fatally, without albuminuria, occurred in my practice :—

J. K., æt. 8, was admitted into the Royal Infirmary, April, 1864, afflicted with general anasarca. She had had scarlet fever four months before, and during convalescence therefrom (in the third week), was suddenly seized with general swelling of the body, which has continued since. When admitted, she presented a perfect type of scarlatinal dropsy—universal and great anasarca, difficultly pitting on pressure, puffy pasty face, excessive pallor of the surface, shortness of breath. On examining the urine not a particle of albumen could be detected, nor any casts or other renal derivatives ; it was scanty and high-coloured. The skin was very dry, and a constant degree of feverishness existed.

She remained under observation until her death, four weeks after admission. The œdema remained stationary ; the urine was repeatedly examined, but never found to contain albumen. The feverishness became more intense, the tongue became dry, and the breath very short ; toward the close there was diarrhœa, which helped to carry her off.

Autopsy, 24 hours after death. Several deep and old tuberculous ulcers were found in the small *intestines*. A few nodules of tubercle, as big as peas, were grouped under the peritoneum, around the bases of these ulcers. The mesenteric glands were enlarged and tuberculous. There was no general tuberculosis of the peritoneum. The *lungs* contained a few old tubercles (of no great size) at the apices. The tuberculous masses were throughout old and inactive.

Both *pleuræ* contained a large quantity of fluid, and the lungs were much compressed thereby.

The *liver* was excessively bloodless. The *heart* was natural.

The *kidneys* were good examples of the "smooth, white" Bright's kidney. They were slightly enlarged, and weighed together 7 ounces. The organs were limp, their surface pale and smooth ; the capsule peeled off readily. The most curious thing about them was, the existence of certain sharply-outlined flat depressions, which differed from the remainder of the superficies. The surface generally was of a characteristic fawn-colour, picked with dead white ; but at these depressed spots the colour was slate-grey, and contrasted markedly, by its blank, grey aspect, with the spotted appearance of the remainder. It was evident that atrophic changes were beginning to take place at these spots. On section, the kidneys presented the usual appearance of the "smooth, white" kidney.

There was no information as to the state of the urine when the anasarca broke out ; but for a month preceding death it was free from albumen, though the general symptoms, and the state of the kidneys after death, bore evidence of the existence of Bright's disease.*

The amount of albumen lost in twenty-four hours varies commonly from 45 to 300 grains ; Dr. Parkes observed in one instance as much as 545 grains. The quantity is larger during digestion than during fasting; it may be twice as great. It rises and falls irregularly in the course of the disease—sometimes diminishing to a trace, and anon increasing to an intense impregnation.

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* Hamilton, out of sixty cases of scarlatinal dropsy observed by him in Edinburgh, encountered two, in which there was no albuminuria.

The urine is generally pale, and not quite clear. It deposits, on standing, an amorphous whitish sediment of renal epithelium and tube-casts. It sometimes contains blood—even in quantity—though generally only in microscopic proportion. When there is intercurrent pyrexia, or the case is complicated with phthisis or regurgitant heart disease, the urine may be high-coloured, and turbid from lithates.

The quantity of urine voided per day varies according to the type of the disease, and the presence or absence of pyrexia, sweating, vomiting or diarrhœa. The urine is throughout scanty with the large white kidney; I have known it not to exceed 35 ounces on any one day for a period of four months, and to be under 20 ounces for three successive weeks, and under 12 ounces for several consecutive days. It may even sink to one or two ounces. With the granular contracting kidney, the urine is abundant (three or four pints a-day) in the middle periods of the disease; but it gradually grows scantier toward the termination; in exceptional instances, the diuresis is profuse, and the urine may occasionally amount to five and even nine pints a day.* These larger quantities have been generally observed after an attack of uræmic convulsions, or coincidently with sudden subsidence of dropsy.

The specific gravity is low when the urine is copious (1006 to 1015); but when it is scanty, the sp. gr. may rise to 1030 or even 1040.

The reaction of the urine is nearly always acid; and, not unfrequently, it deposits uric acid and oxalate of lime. Occasionally I have noted it alkaline from fixed alkali, and twice ammoniacal on emission.

The *renal derivatives* (epithelium and tube-casts) are markedly scantier in the chronic, than in the acute forms of Bright's disease; and it is not uncommon for them to be altogether absent for limited periods. They are, however, sometimes discoverable when the urine has ceased (temporarily) to be albuminous. The appearances of the discharged epithelia and casts present considerable diversities, which supply an important insight into the structural changes going on within the kidney. The epithelial cells may be simply withered; more rarely they are totally disintegrated into an amorphous granular débris; in other cases they contain specks of oil, or they may even be wholly converted into an agglomeration of oily particles so as to appear identical with the so-called "granular corpuscle," or "inflammation globule." The casts are sometimes similarly speckled with fat, and free oily dots are scattered over the field. Such a conjunction indicates a fatal disorganization of the organs—either large fat kidneys, or contracted granular ones. But the casts *most commonly* seen in chronic Bright's disease are "small" and "large" hyaline forms, and "granular" opaque ones. Any of these may have a few wasted epithelial cells strewed over them. Perfect "epithelial" casts are rare in chronic cases, and blood casts are still more rare, unless there be concomitant tricuspid regurgitation.

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* Christison, pp. 174 and 186. Pfeufer, in Henle and Pfeufer's Zeitsch., Bd. I., p. 58.

When intercurrent exacerbations of the renal process, with pyrexia, arise, there will be found (whatever may have been the previous character of the casts) medium-sized and large solid-looking, pale-straw, albuminous casts resembling molten glass (see Fig. 45).

Casts of these diverse appearances may be discharged by the same individual, even during the same day. Conclusions as to the probable state of the kidney can only be drawn from the *prevailing* character of the deposit, and not from one or two individual casts or cells. This diversity in the character of the casts arises from the different condition of the several parts of the gland. In some portions the tubuli may be denuded of their epithelium, and the exudation thrown into them is discharged in the form of large hyaline casts; if the denuded portions have undergone subsequent contraction the casts will be small and hyaline. Other tubes, clothed or partially clothed with epithelium, shed some of their cells with the contained exudation, and cause the appearance in the urine of casts more or less studded with epithelial remnants.

The longer the exudation is retained within the tubuli, the darker and more granular will it appear, when discharged as casts; and *vice versâ*, casts speedily discharged are commonly hyaline. Sometimes casts are darkened by the colouring matter of the blood; and the opaque granular ones are (sometimes at least) composed of crushed epithelial débris moulded into the form of the tubuli. (See *Diagnosis.*)

The normal solids of the urine are all diminished in chronic Bright's disease. The urea is, as a rule, markedly reduced—the daily quantity averaging only about 100 grains; Frerichs has observed it as low as 15 grains.* There is no correspondence, direct or inverse, between the excretion of urea and the discharge of albumen. With intercurrent pyrexia the excretion of urea rises.

The changes in the *blood* are the complement of those in the urine. The blood becomes more watery and poorer in albumen and red corpuscles. On the other hand urea, uric acid, the extractive matters and the pale corpuscles accumulate in it. This alteration in the composition of the blood is deeply concerned in the production of the more prominent features of the disease—the anæmia, dropsical effusions, uræmic phenomena and secondary inflammations.

Dropsy is much oftener absent in the chronic than in the acute form. It is much more constant with the smooth large, than with the granular contracted kidney. Of the latter class probably one-third or one-fourth of the cases run their entire course without dropsy. The effusion begins quite as often in the feet and legs as in the face; it is commonly slight and partial, but sometimes excessive and general. When the heart or liver is diseased, ascites and œdema of the legs become disproportionately prominent. The effusion is apt to change its seat capriciously; and it comes and goes from time to time. Sometimes it disappears totally for months, and then returns again. More frequently, after a subsidence of the general dropsy, œdema lingers obstinately in one or two places—over the flat of the tibiæ, about the ankles, beneath the eyelids, under the conjunctival membrane, or about the genitals. The presence or absence of dropsy, generally, but by no means always, corresponds with the abundance or scantiness of the urine; but it has no relation to the amount of albumen.

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* Exceptions occur to this rule. Mosler mentions a case of Bright's disease in which 640 grains of urea were voided in one day (Archiv d. Vereins, Bd. xi. p. 513). Schottin found creatine and creatinine increased in the urine in Bright's disease, and the increase was observed to keep pace with the intensity of the uræmic symptoms (Archiv der Heilk., 1860, p. 417).

The *skin* is usually obstinately dry; perspiration is quite exceptional; and when it occurs, is commonly due to diaphoretic measures of treatment. Profuse sweating does, however, sometimes take place spontaneously, and may even continue for weeks. In one such case under my care an abundant crop of pemphigus vesicles broke out on the surface. The integuments in some cases are excessively pale and glossy, but more commonly they are sallow and rough. There is little or no tenderness in the renal region in the chronic cases, and the frequency of micturition is mostly observed at night.

Some degree of *bronchitis* is almost an invariable coincident of Bright's disease both in the acute and chronic form.

Complications and connection with other diseases. The digestive organs are nearly always disturbed: at first there is loss of appetite and nausea; in the later periods frequent or even uncontrollable vomiting is not uncommon. The bowels are alternately bound and loose. Severe fitful diarrhœa, which leaves the dropsy undiminished, is not uncommon, especially toward the close of the complaint. Not unfrequently, anatomical lesions are found in the *intestines* which explain these disturbances; in other cases they are manifestly uræmic. Treitz states that urea is discharged into the intestines from the blood, and converted into carbonate of ammonia, which acts as an irritant on the intestinal mucous membrane. The more palpable changes found in the intestines are,—follicular catarrh, dysenteric ulcers—sometimes with sloughing of the mucous membrane. In 220 cases of Bright's disease collected by Treitz, the following conditions of the intestines were found after death.*

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* Prag. Vierteljahrschr., 1859.

Hydrorrhœa (intestines filled with yellow-greenish fluid)	80 times.
Blennorrhœa and catarrh	60 ,,
Croupous and ulcerous dysentery	19 ,,
Sloughing	12 ,,
Sanguineous contents without discoverable source of hæmorrhage	4 ,,
Normal fæces	5 ,,
Contents of intestines undetermined	11 ,,

Secondary inflammations of the lungs, endocardium, pericardium, pleura, peritoneum or integuments, may break out at any period in the course of chronic Bright's disease. The tendency to these constitutes one of the principal dangers of the complaint. Cardiac hypertrophy, valvular disease, and pulmonary tubercle are frequent complications.

The following table exhibits the proportionate frequency with which the various organs, other than the kidneys, are found affected in Bright's disease generally. It contains the results of 406 autopsies, contributed as follows:—Bright, 100; Christison, 14; Gregory, 37; Martin-Solon, 8; Rayer, 48; Becquerel, 45; Bright and Barlow, 10; Malmsten, 9; Frerichs, 21; Rosenstein, 114.

Heart.	Lungs.	Pleura.	Pericardium.	Peritoneum.	Liver.	Spleen.	Stomach and Intestines.	Brain.
125 times hypertrophy; 54 times with valvular disease; 55 times without valvular disease.	115 times œdema of lungs; 52 pneumonia; 8 pulmonary apoplexy; 4 gangrene; 37 tubercle; 33 vesicular emphysema.	57 pleurisy.	30 pericarditis.	46 peritonitis.	41 cirrhosis; 38 fatty liver.	58 chronic tumour; 17 acute splenic tumour.	36 gastric catarrh; 85 catarrh and follicular ulceration of intestine; 13 tuberculosis of intestine.	14 sanguineous apoplexy; 59 effusion of serum under arachnoid.

In addition, there were:—1 case complicated with cancer of the liver; 4 cases with cancer of the pylorus; 2 typhoid ulcers of intestines; 2 meningitis; 1 meningeal tubercle; 11 tumor cerebri; 3 abscess of lung; 11 nutmeg liver; 3 lardaceous liver; 9 contraction of spleen; 3 diphtheritis of intestines; 1 softening of brain; 6 chronic arachnitis; 1 suppurative meningitis.

Bright's disease and phthisis. Phthisis is a frequent complication of Bright's disease. Eight cases have fallen under my observation in the last two years. In five of these, the pulmonary disease was clearly antecedent in point of origin to the renal; in three the sequence was the reverse. The co-existence of two fatally-tending diseases might have been expected to accelerate the inevitable issue; yet most cases of this class run an exceedingly chronic course, and continue in a stagnant condition for months together. In the following remarkable instance, the pulmonary disease (already in its third stage) almost completely retrograded, and was supplanted by the renal affection:—

M. C., æt. 20, a mechanic, was admitted into the Royal Infirmary, Oct. 27, 1863. He was a well-grown young man, with white pallid features, dry skin, heavy eyes, and moderate œdema of the lower extremities. The abdomen was enlarged from ascites, and the integuments of the flanks and hypogastrium were œdematous; pulse 112, regular, small; respirations 21; tongue moist, slightly furred. The state of the chest on admission was as follows:—Diminished expansion over both apices; but more on the right side than the left; conspicuous depression of the right infra-clavicular region. There was almost complete loss of resonance on the right side as low as the second interspace. The right upper scapular regions were also dull on percussion. Moist crepitation and cavernous rhonchi were heard beneath the clavicle on both sides. Whispering pectoriloquy was very distinct below the right clavicle, and present, though less typically, over the left apex. The heart's sounds were natural; there was no appreciable hypertrophy. The expectoration was copious, airless, purulent.

The urine was scanty, amber-coloured, spec. grav. 1030, intensely albuminous, becoming almost solid on boiling. A slight deposit of withered renal epithelia and transparent tube-casts without any, or only very faint, signs of fatty changes, lay at the bottom of the glass.

The history disclosed perfect health until ten months ago, when the patient began to cough. He attributed these symptoms to cold taken by passing out into the cold air from his hot work-room. His family is tuberculous: a sister came subsequently under my care with phthisis. He had night perspirations six months ago. Three weeks before admission the ankles began to swell; but the skin had been dry for three months.

Cod-liver oil and iron were prescribed; a warm bath was administered every other evening. The patient constantly kept his bed, on account of the swelling in his legs increasing when he sat up.

For a period of two months I was unable to attend at the Infirmary on account of illness, but the treatment was carried on during my absence without alteration, and the patient kept continuously in bed. When I revisited the wards in Jan. 1865, I found the renal symptoms somewhat advanced; but the pulmonary complaint had decidedly receded. The urine was very scanty, varying from 12 to 18 and 26 ounces a day, with a specific gravity ranging from 1030 to 1034; it often deposited amorphous urates. On Jan. 29th the urinary deposit corresponded to the following description:—It was scanty, and composed of atrophied renal cells, with a few excessively transparent small hyaline casts, some of which were speckled with albuminous granules and a few doubtful oil particles (see Fig. 44). The patient at this date was in a quiescent state and free from fever.

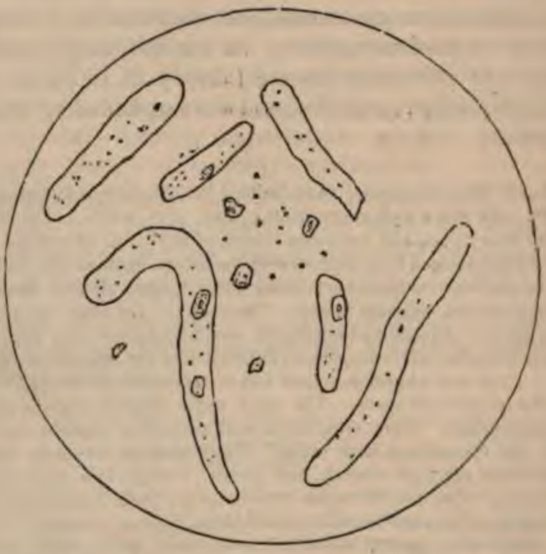

FIG. 44. Transparent hyaline casts, from the urine of M. C., on Jan. 29th (quiescent period).

The chest complaint was now altogether in the background; there was scarcely any expectoration, and the physical signs indicated a marked amelioration. The depression under the right clavicle was less conspicuous, and the movement improved; the percussion sounds were still unaltered, and the rhonchi still cavernous, but not abundant; pulse varied from 88 to 100; resp. from 20 to 22.

During February the urine became still scantier (12 to 20 ounces a day), with a density ranging from 1033 to 1041. It became almost solid on boiling. The anasarca increased, and extended into the face and upper limbs. Occasional vomiting took place, and the appetite failed entirely.

In the last week of February, the patient insisted on going home. But he had not been out a single day before he took a violent cold, ushered-in with repeated shiverings. The anasarca increased rapidly; respiration became oppressed, and he was re-admitted three days later (Feb 26th) in the following state :—Great general dropsy, the urine almost suppressed, distressing oppression of breathing. A compound jalap powder was administered ; after which he vomited and had three loose motions.

On the following day, the patient was very thirsty and feverish ; tongue furred, red at edges ; pulse 128 ; resp. 30 ; cough very distressing ; a scanty expectoration of nummular purulent sputa. He complained loudly of pains in the abdomen, chest, and back, especially when he turned in bed. There was great restlessness.

A hot-air bath was administered, with the effect of inducing copious sweating, and reducing the œdema somewhat.

Feb. 28th.—Urine, last 24 hours, only 8 ounces ; it was intensely albuminous and deposited urates. The renal derivatives presented totally new characters ; they are delineated in Fig. 45 ; the new feature was the appearance of massive molten-looking casts of large and medium size. Some of them were slightly granular in spots ; a few were also sparsely studded with epithelium ; but there were no proper "epithelial casts ;" there was neither blood nor fat. Some of the large casts lay side by side like thick logs, and appeared as if split in a longitudinal direction at their extremities (Fig. 45).

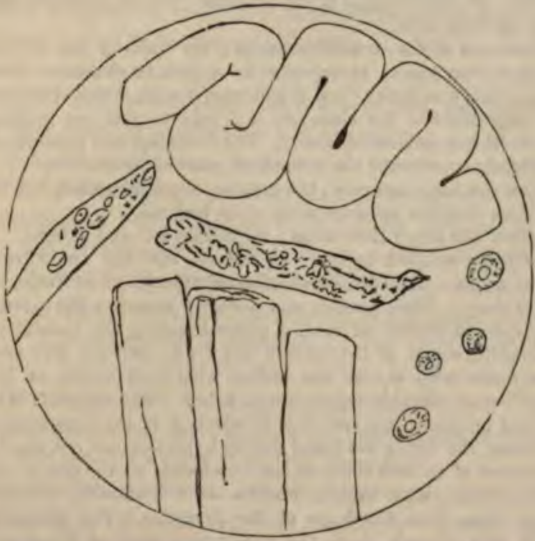

FIG. 45. Massive molten-looking casts, from the urine of M. C., on Feb. 28 (pyrexial period).

On the 28th the patient grew feebler and more restless ; obstinate vomiting set in, and continued nearly till death, which took place on the morning of the 29th. Only two ounces of urine were passed in the last

24 hours of life. There were neither convulsions nor coma; and vision continued good to the last.

Autopsy, 30 hours after death. The *right lung* presented an exquisite example of retrograde phthisis. Half-a-dozen small cavities were counted in the upper lobe—all of them small, varying from the size of a pea to that of a horse-bean—completely lined with a thick pyogenic membrane. Not a particle of tubercle existed around these cavities nor in any part of this lobe. The pulmonary tissue was dark and leathery, and very imperfectly aërated. The right apex was condensed, deeply puckered, and traversed in various directions by thick white lines of cicatricial tissue. In the lower lobe of the same lung, a vomica as large as a filbert was found with anfractuous boundaries composed of tuberculous matter. Small masses of obsolete tubercle—some cretaceous, others putty-like—were scattered sparsely through the lower lobe.

The *left lung* was crepitant throughout. The upper lobe contained three cavities—one as big as a walnut—lined with pyogenic membrane, and not surrounded by tubercle. Small nodules of tubercle were scattered through the upper and middle lobes—some cretaceous, some putty-like, others unsoftened and crude. The inferior lobe of the left lung was highly œdematous. No fluid existed in either pleura; but old adhesions prevailed sparingly on both sides.

The *heart* was of the usual dimensions; the walls of the left ventricle were thicker than was to be expected in a case of phthisis. Both sides were filled with firm bulky clots of yellowish fibrine, which closely adhered to the inequalities of the chamber, and sent voluminous prolongations into the aorta and pulmonary artery. The formation and presence of these clots evidently constituted the immediate cause of dissolution.

The *liver* was large and pale; the hepatic cells well-formed, and not containing more than the usual quantity of fat molecules.

The *spleen* was larger than usual; its texture soft and natural.

The *kidneys* weighed together 23 ounces, and the two were almost exactly of a size. They furnished a typical example of the large, smooth, mottled kidney. Their surface was perfectly smooth; the capsule, thin and transparent, peeled off readily without tearing the glandular tissue. The prevailing colour of the surface was fawn, marbled here and there with red; the fawn colour was picked with dead white, as in ivory. The organs were conspicuously soft and flabby. The red parts of the surface showed minute spotty and sinuous injection of the superficial vessels.

On section, the cortex was found greatly hypertrophied; it stood half or three-quarters of an inch thick on the broad ends of the cones. It had a full fawn colour, with broken streaks of red running through it in diverging lines, from the bases of the pyramids. The pyramids were unusually pale, though, from the ex-sanguine state of the cortex, they offered a pretty strong contrast of colour with the latter. The epithelial lining of the convoluted tubes was extensively disorganised; both cell and nucleus were reduced to a granular, fatty débris. Scarcely a single cell approaching perfection could be seen. The Malpighian corpuscles were not altered in size; but they were penetrated, and rendered opaque, by a granular material. The epithelium of the straight tubes was in much better preservation, not only the nuclei could be seen, but the outlines of the cells themselves. A considerable quantity of spindle-shaped fibre-cells were found, also medium-sized massive-looking casts—resembling those found in the urine shortly before death.

Two things appeared singular in relation to these kidneys, namely, that the urine should contain so few renal derivatives when the kidney contained such an immense quantity in a disorganised state; and secondly, that this disorganised material should contain fat in such quantity without there having been any, or scarcely any, in the urinary deposit. Perhaps that the degeneration of the epithelium only attained this maximum degree in that last intercurrent febrile attack which immediately preceded death; and that the plugging up of the canals of the pyramids with the massive casts, which then appeared for the first time in the urine, was the determining condition of the suppression of urine, and of the absence from it of the derivatives of the convoluted tubes.

It is quite uncertain what fundamental connection there may be between phthisis and Bright's disease. Bright himself thought there was a degree of antagonism; and he pointed, in support of his opinion, to the fact, that patients with degenerated kidneys do not often become phthisical; but this may be owing to the more advanced age of the great majority of sufferers from chronic Bright's disease. In young persons the coincidence of the two diseases is certainly not uncommon. Yet the foregoing instance would seem to show, in a striking manner, that chronic Bright's disease is, at least, not especially favourable to the deposition of tubercle; and it might be questioned even, whether the retrocession of the tubercle would have become so complete had the kidneys remained healthy. On the other hand, it might be argued that the retrocession of the tubercle was not owing to the antagonistic influence of the renal disease, but was due to the long maintenance of the body at rest and in an equable temperature by the prolonged lying-a-bed.

Bright's disease and heart disease. The connection of cardiac disease with renal disorder is at least threefold.

In the *first* class of cases, simple *hypertrophy* of the heart, and especially of the left ventricle, is found without valvular incompetency and without degeneration of the muscular fibres. In this class, which is a numerous one, as the table at p. 340 shows, the cardiac affection is secondary to the renal. Bright, who was the first to point out this curious coincidence,* offered two explanations of it—either, that the altered composition of the blood exercised an irregular and unwonted stimulation upon the muscular tissue of the heart, or, so impeded the circulation in the capillaries that a greater effort of the ventricle was required to drive the blood through the distant minute branches of the blood-vessels. Traube[*] explains the occurrence of simple cardiac hypertrophy in chronic Bright's disease somewhat differently. In his experience the kidneys in these cases are markedly atrophied.[†] The contraction of the renal tissue involves destruction of a certain amount of secreting structure and a diminution of the flow of blood through the organs. Two consequences follow, namely, that a diminished amount of blood passes from the arterial into the venous system, and that a less quantity of fluid is withdrawn from the arterial system for the formation of urine. Both circumstances, but especially the second, operate to increase the tension in the arterial system, and consequently to increase the resistance which the left ventricle has to overcome in discharging its contents. The hypertrophy which follows is, therefore, according to Traube, a conservative or compensating change, similar, in the mechanism of its production, to that induced by valvular incompetency or aortic constriction. If the compensation be complete, the heightened tension in the arterial system occasions a larger transudation of water and even of urea and other urinary solids, through the kidneys, and in that manner materially helps to stave off dropsical effusion and uræmic symptoms. But should some additional obstruction to the circulation arise, through intercurrent inflammation of the bronchial tubes, or of the lungs, pleura, or pericardium, the heart—enlarged and strengthened though it be—no longer suffices to overcome the increased resistance, and dropsical effusions or uræmia speedily make their appearance. Traube adduces some apposite examples in which individuals with contracted kidneys enjoyed fair health, with capability of exertion, and continued free from anasarca and uræmic disturbance, until the advent of some complication disordered the balance of the circulation, and *then* the urine became scanty, and the familiar symptoms of renal disease, previously latent, broke forth into prominence.

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* Guy's Hospital Reports, vol. i. p. 396.

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[*] Ueber den Zusammenhang von Herz-und Nieren-Krankheiten, p. 58.

[†] In a later communication Traube brings forward evidence to show that hypertrophy of the left ventricle is an almost constant concomitant of granular and contracted kidney. In 77 cases collected by him from various sources, the left ventricle was found hypertrophied in 93 per cent. (*Deutsche Klinik*, 1859, p. 315). Förster, in a short paper on this subject in the *Würzburger Med. Zeitschr.* for 1864, publishes some facts (inconclusive, as it appears to me) which are adverse to Traube's views.

In the *second* class of cases, *valvular* defects and their consequences co-exist with Bright's disease. Most of these are examples of endocarditis, secondary to the renal disease. But in other cases the cardiac and renal affections arise independently of each other, and depend on some cause common to both—as in the following example :—

J. H., æt. 48, was admitted into the Manch. Roy. Infirmary, Nov. 22, 1862. He had right hemiplegia : the mental faculties were wholly disordered ; there was gay incoherence and insanity ; no fever. The heart's apex beat in the fifth interspace, half an inch outside the nipple line ; a loud systolic bruit was audible at the apex and the mid-sternal base, and extended up the aorta ; a faint diastolic bruit was audible over the second right cartilage. The urine was albuminous to about one-fifth : there was no dropsy.

The patient remained in the Infirmary a month. The mental derangement subsided in ten days and perfect coherence returned. The other symptoms remained unchanged. He returned home ; and in a few days was seized with coma, which proved rapidly fatal. The autopsy revealed —granular red kidneys with abundant presence of fat ; cardiac hypertrophy with fatty degeneration of the muscular fibres ; extensive disease of the mitral and aortic valves with atheromatous patches on the aorta : two old apoplectic clots were found in the left hemisphere, and wide-spread fatty degeneration of the arteries existed at the base of the brain.

In this instance fatty degeneration had simultaneously invaded the heart, the brain, and the kidneys, and produced a triple series of symptoms —all essentially independent of each other.

The *third* class of cases are those in which the renal disorder (congestion, &c.) is secondary and subordinate to cardiac disease. These cases have already been fully described in Chap. I. in connection with CONGESTION OF THE KIDNEYS, which see.

URÆMIA.

Certain phenomena, chiefly affecting the nervo-muscular system, arising in the course of Bright's disease, have been attributed to a poisoned state of the blood, from the retention in it of excrementitious matters which the disabled kidneys are unable properly to eliminate. To these phenomena the term *uræmic* has been applied ; they consist of twitchings and convulsions of the voluntary muscles, headache, drowsiness coma, defects of sight and hearing, vomiting and diarrhœa.

It is a marked feature of uræmic phenomena that those which are of a paralytic nature affect the sensorium and the special senses, but not the voluntary muscles ; while those of an opposite kind (exalted irritability) affect the voluntary muscles, but not the sensorium. Delirium is rare, while coma is frequent ; paralysis of the limbs is scarcely known (unless there be some anatomical lesion of the brain superadded), while convulsions are frequent.

The mode in which uræmic symptoms enter on the scene, and the forms they assume, present great diversity.

Generally they begin insidiously with headache or vomiting, followed by heaviness, indifference, and somnolence. These premonitories may either pass away in a few days without further consequence, or they may be succeeded by general convulsions and coma. In other instances the patient is at once struck down with convulsions or insensibility without any previous warning, or he becomes suddenly blind, or is seized with uncontrollable vomiting.

The most common of these symptoms is *headache ;* few individuals with degenerated kidneys altogether escape it. A sense of heavy weight or compression is complained of over the forehead or vertex. Sometimes the pain is obstinately fixed at the back of the neck, or behind the orbits.

The *defects of sight* consist either in a dimness of vision (amblyopia), which comes and goes—objects appearing as if veiled in mist ; or in rapid and complete, though usually temporary, blindness. The convulsive seizures are often accompanied with temporary loss of sight, which generally persists in greater or less degree for a certain time after the spasms have passed away.

The ophthalmoscope reveals no organic change in the eye in genuinely uræmic amblyopia ; it is a purely cerebral phenomenon, and not to be confounded with the hæmorrhagic blindness (Retinitis apoplectica), which is also not uncommon in Bright's disease, and which is due, as V. Graefe has shown, to rupture of the retinal vessels. In this latter affection (which is in no sense uræmic) the loss of sight is seldom complete, but is of a more permanent character. The production of it is probably due to the hypertrophy of the left ventricle which so commonly accompanies a contracting kidney, and the increased tension in the arterial system consequent thereupon; it is an occurrence of the same order as the sanguineous apoplexy to which the same individuals are liable.

Uræmic *deafness* is much less common than amblyopia, and its occurrence is highly exceptional.

Uræmic *convulsions* are of the epileptic type, and, as a rule, they conform strictly to that type—being accompanied with complete insensibility, rolling of the eyes, biting of the tongue, and foaming at the mouth. The paroxysm commonly leaves the patient deeply comatose.

In exceptional instances consciousness is not wholly lost. In a lady under my care the paroxysms coincided with the catamenial periods; during the convulsions the patient knew the persons about her, and called loudly to be held fast. A case is related by Bright in which the spasms at first resembled cramps; these were followed by twitchings of the hands, arms, shoulders, chest, and legs. The spasms were almost constant, and caused a somewhat hurried mode of expression when the patient spoke, but the intelligence was perfect. As the case proceeded the spasms became more and more severe, with forcible drawing up of the legs, and distortion of the muscles of the face; the faculties were retained to the last.*

An attack of uræmic convulsions may consist of only a single paroxysm; more frequently there occur a succession of paroxysms or fits, following each other at uncertain intervals of a few minutes or several hours—the patient lying during the remissions in a state of profound insensibility, with stertorous breathing, pale face, and dilated pupils; or in deep drowsiness, but capable of being partially roused, when spoken to or shaken.

If a first attack does not prove fatal, it may recur at irregular intervals of weeks or months, or be replaced by uræmic symptoms of some other order.

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* Guy's Hospital Reports, 1840, p. 139.

Uræmic *coma* either creeps on very gradually, passing on, in the course of two or three days, into complete stupor; or it culminates quickly—the patient falling down, as if in apoplexy, perhaps while walking in the streets, or occupied with his usual avocations. Cases of this class, when there is no anasarca, and the previous state of the urine is unknown, are very liable to be confounded with apoplexy or with narcotic poisoning. The following instructive illustrations of such an occurrence are related by Mr. Moore and Dr. Richardson:—

Case I.—An old soldier, named Price, was received into Queen's Hospital, Birmingham, with the following symptoms:—breathing laborious and sometimes stertorous; when left alone the patient passes into a state of stupor, answers questions sensibly when roused; pupils moderately dilated, indolently sensitive to light.

It appeared that Price, having suffered some days from diarrhœa, went into a druggist's shop and asked for a pennyworth of tincture of rhubarb. The shopman added to this dose a few drops from another bottle, and Price swallowed the whole before leaving the shop.

Immediately after taking the above dose he became drowsy and vomited; at the suggestion of a neighbour he returned to the shop, and asked the shopman whether he had given him laudanum. The latter told him that he had put in a few drops on account of the severity of his symptoms. Upon again reaching home he fell asleep, and continued sleeping unless temporarily roused. In this state he was taken into hospital, and was treated as a case of opium poisoning. A mustard emetic was ordered immediately; the patient was kept in constant motion, and plied with strong coffee. He improved considerably under the treatment, and talked over his old campaigns with the porter who had charge of him. Next day he relapsed into a lethargic state; galvanism was employed without benefit; he was now walked round the hospital-garden between two men, and strong infusion of green tea was administered. A little improvement followed, but at 2½ p.m. he relapsed once more, and the breathing became more oppressed. As long as he was kept moving he could be made to answer questions; but in the course of the afternoon the somnolence deepened in spite of the treatment. Ether and ammonia were applied to the nostrils; cold water was dashed over the face and neck; but at 8 p.m. the drowsiness had become insuperable; the stertor augmented. Mustard poultices were put to the legs. Venesection was tried, but when four ounces of blood had flowed the pulse became thread-like, and it was thought prudent to desist. The coma increased in intensity; and he died at 2.30 a.m., 44 hours after admission, and 102 hours from the time of taking the dose.

A coroner's inquest was held on the case, on account of the suspicion of poisoning; but the results of the *post mortem* went to exonerate the druggist, for the kidneys were found granular and greatly atrophied, and the urine left in the bladder was found albuminous. (J. Moore, *London Med. Gaz.*, 1845, p. 821.)

CASE II.—A woman, aged 34 years, who was given to drinking, and had recently been treated for primary syphilis, was seized on Nov. 18th, 1859, with rigors. She was attended by a neighbouring chemist, who, on Nov. 22nd, gave her a mixture which, he said, contained dilute nitric acid, nitrate of potassa, syrup of buckthorn, sulphate of magnesia, and water. It was afterwards proved by analysis that these were the constituent parts of the remaining portion of the mixture. The medicine was sent in to the woman on the evening of the 22nd; and after taking a large quantity of beef-tea, she swallowed one dose of the mixture. Five minutes afterwards she became hysterical and convulsed, and the friends believing the woman to have been poisoned, summoned the druggist, who in alarm tried to get her to take some ipecacuanha, but without avail. In the course of the night a medical man was called in; he found the woman in a state of typhoid coma, with pupils slightly dilated and immovable, and the body at times convulsed—the convulsions assuming an epileptiform type. Every available means of treatment was carried out, but the coma became more profound, and seventy hours after the administration of the mixture above mentioned, death closed the scene. By the coroner's warrant the body was examined, and a chemical inquiry instituted. The brain was quite healthy; the kidneys were greatly diseased—large, flabby, pale, speckled, soft, and greasy. The analysis disclosed no poison of any sort. (Richardson, *Clinical Essays*, p. 135.)

CASE III.—A gentleman, æt. 63, was driving in an open chaise through the village of Mortlake, in 1853; he was observed by his servant, who was by his side, to be constantly drowsy; at last he suddenly seemed to fall into a helpless state and dropped from the chaise. He was conveyed into a house, and Dr. Richardson was summoned. Dr. R. found him suffering from all the signs of narcotic poisoning; the pupils were fixed and slightly dilated. Some urine was withdrawn from the bladder and found to be largely charged with albumen. He recovered from the attack; but three weeks later he suffered again in the same way and died with typhoid coma—the urine being altogether suppressed for many hours before death, and having been albuminous throughout the illness. (Richardson, *Clinical Essays*, p. 141.)

The *diagnosis* of uræmic coma from apoplexy rests on the absence (in the former) of paralysis, and the partial recovery of consciousness between the convulsive attacks—if there be any. From poisoning by opium, renal coma is distinguished by the dilated or semi-dilated state of the pupils, and by the occurrence of remissions in the insensibility. From ordinary epilepsy the diagnosis—apart from the antecedent history, which, if known, suffices to indicate the nature of the case—is sometimes difficult. The incidents of the seizures are often identical, even to the existence of an aura. As a rule uræmic fits want the turgid purplish countenance and asphyxial character of true epilepsy — the face in uræmia being nearly always deadly pale and the breathing easy.

Dr. Richardson relates the cases of two children poisoned by belladonna berries, in which the symptoms closely resembled uræmic coma sequential to scarlatina. The insensibility was complete, and the pupils strongly dilated. The examination of the vomited matters and of the urine furnishes, in such cases, the best means of diagnosis.

In all cases of convulsions or insensibility from doubtful causes, the urine should be forthwith examined, and, if necessary, withdrawn by catheter for that purpose. It must not, of course, be forgotten, that sanguineous apoplexy is a not very unfrequent occurrence in chronic Bright's disease, as in the case of J. H. before related (p. 347).

Uræmic coma and convulsions may prevail separately : but much more commonly the attacks are of a mixed character, and combine several or all the phenomena just enumerated. The varied character and fitful course of uræmia are graphically illustrated in the following accurately-observed example, by Dr. Liebermeister (Prag. Vierteljahrschr., Jahrg. xviii., Bd. iv.) :—

Wilhelmina Karsten, a servant-maid from the country, æt. 29, was admitted into the Greifswald Hospital on June 8th, 1859.

Previous History.—The patient, according to her own statement, was perfectly healthy in her youth. In her eighteenth year she suffered for six weeks from tertian ague. Three years ago she became pregnant : toward the end of her time, œdema of the lower limbs set in, which, however, soon after the birth of a healthy child, disappeared. During the two following years she continued in good health, and capable of pursuing her employment. In the first week of November, 1858, the patient noticed, without previous signs of disease, slight œdema of the ankles, which gradually increased, and extended to the hands, face, and abdomen. These swellings sometimes increased, sometimes diminished, but never entirely subsided, and latterly they steadily progressed. At first the patient was able to continue her work ; but she remarked that any unusual exertion produced considerable shortness of breath, severe palpitation, and a feeling of increased heat. Headaches, which had troubled her occasionally at the beginning of her illness, became more frequent and more severe during the winter, and were conjoined with fugitive disorder of vision, great weariness, and sleepiness. These attacks were never accompanied with vomiting. The appetite was constantly poor. During the winter, diarrhœa often occurred without assignable cause, but was not of long continuance, nor did it recur in the last few months. Occasionally there was cough with scanty expectoration. The urine does not seem to have been at any time markedly deficient; at times it was even more abundant than natural. Menstruation, at first regular, had not occurred for three months.

Present State.—The patient was of middle height, moderately strongly made; the countenance, the cutaneous surface, and the visible mucous membranes, were markedly pale. The face was slightly œdematous; the upper limbs and the integuments of the chest and back, strongly so. The legs were highly anasarcous, the labia majora and abdominal parietes greatly swelled, and there was considerable ascites. Hepatic dulness reached from the nipple almost to the borders of the ribs. The cardiac dulness was not increased in breadth, and the sounds were pure and loud. The second sound was especially loud over the pulmonary artery. The pulse was of normal frequency, full, hard, and incompressible. There were signs of hydrothorax and bronchial catarrh. The urine was of natural quantity, pale, albuminous, somewhat turbid, with an abundant sediment. The sediment consisted of vaginal epithelium, pus, and casts of tubes of various thickness, partly hyaline, structureless, or somewhat twisted and folded, partly beset with numerous dark, sharply-defined, very minute granules. The appetite somewhat bad; bowels opened daily.

The patient was put on a nutritious diet, and Tinct. ferri Acetatis. As the dropsy did not diminish, Acetum Scillæ in saturation was substituted on June 16th; and as this again proved inefficient to produce diuresis, iron preparations were resumed.

On the night of the 22nd of June, the patient suffered from severe headache; on the 23rd, this passed away, but returned again at mid-day on the 24th. On the same day the sight of the right eye became somewhat affected: and there was great apathy and drowsiness. No nausea or vomiting. On the evening of the 25th, the headache vanished; but the patient continued to see worse with the right than with the left eye for several days.

These slight uræmic phenomena thus passed away, and a general improvement set in; the dropsical effusions also underwent a slow diminution.

On the 15th of June, moderate diarrhœa set in, without exercising much influence on the anasarca, which rather increased. An effort was therefore made to reduce the dropsy by diaphoretic measures. A hot bath was administered on July 5th, and afterwards the patient was wrapped in blankets. Copious sweating followed. The patient slept well until three in the morning, when a violent headache arose and continued for three hours. In the morning the face was found more œdematous, hot, and red; and the patient complained of a severe sense of heat in the head. Pulse 72. After a slight interruption, the use of the baths was continued, and with favourable effects. The anasarca steadily diminished; by the 30th of July it was confined to the more dependent parts of the body. The oppression of the breathing was quite gone; and the appetite considerably improved. On the night of the 10th of August, there occurred severe headache, vomiting twice, and watery stools thrice. The headache continued next day without fever, and the patient was sunk in deep apathy; when spoken to she only opened her eyes. On the day following these symptoms passed away; but the headache returned from time to time. On the night of the 21st of August, severe headache recurred, and persisted through the ensuing day; in the evening the patient complained of buzzing in the ears and sparks before the eyes. Pulse was frequent. On the following night, about two in the morning, a violent paroxysm of general convulsions occurred, of an exactly epileptic character. Consciousness was completely abolished; clonic cramps of the extremities, face, and trunk followed, which were of such severity that the bed shook. In about ten minutes the paroxysm ceased, and the patient lay quiet, foaming at the mouth, with frequent pulse and stertorous breathing. When called, she opened her eyes, stared at the person addressing her, or rolled her eyes hither and thither. When questioned, she gave no answer, did not protrude the tongue when asked to do so, nor made any other movement. The paroxysms, with intervening pauses, in which consciousness did not return, were repeated during the night. By the morning, about twelve fits had thus followed each other. At eight o'clock in the morning consciousness had returned, but the patient was in a highly apathetic state. She complained, when questioned, of headache; vision was greatly weakened. At nine another fit occurred, and by midday three more had taken place. The patient passed urine and fæces under her; and lay, in the pauses, with open mouth, half-shut eyes, and stertorous breathing, fully comatose. Pulse 114-120. In the afternoon no fit occurred; in the evening the patient was still somnolent, but completely conscious: pulse 96. The face was intensely œdematous; in other parts the œdema had undergone no change. Next morning she complained of great weakness and prostration, so that movements were only accomplished with much effort and pain. The organs of sense were no longer disturbed, and the pains in the head less intense. On the following day there was amendment, but still the sense of fatigue, prostration, and apathy, continued. The condition then became changeable; the headache sometimes vanished altogether. Nevertheless, and in spite of abundant secretion of urine, the dropsy increased considerably.

On the 4th of October there occurred a slight rigor and severe headache; during the afternoon, great dyspnœa set-in, with a high degree of somnolence. At 3 p.m. six ounces of blood were withdrawn by venesection. At 6 p.m. there was vomiting, which recurred several times; and about nine, a violent eclamptic attack occurred, which resembled the previous ones, and was followed by coma. The fits returned during the night and the following day about twelve times. The pupil, during the paroxysms, was neither dilated nor contracted, it was only feebly sensitive to light. The administration of calomel and jalap occasioned copious stools: and on the morning of the 6th the patient was conscious, the face very œdematous; she complained of headache, and was highly apathetic. On the 7th and 8th the facial œdema still increased, and the arms were intensely anasarcous. The ascites and œdema of the feet appeared slightly diminished. Slow improvement then took place. On the 22nd threatening symptoms again appeared, the pains in the head became intense, somnolence ensued, and, from time to time, twitchings of the upper extremities. Again all these passed over: but on the afternoon of the 31st there was a slight fit of convulsions, followed by vomiting twice; at 5 p.m. an eclamptic paroxysm of great severity occurred, and two more during the night. In the intervals between the convulsive paroxysms the patient lay completely insensible, with open mouth, stertorous breathing, and cyanotic countenance. The teeth were covered with sordes. Both pupils were strongly contracted. Next day the patient was again conscious, but dull and apathetic. The countenance was very œdematous; pupils sensitive to light. This sleepy indifferent state continued through the ensuing week.

From the 10th of November the visual power showed great disturbance, and the condition changed about. Sometimes the patient could easily count fingers, sometimes she could do so only imperfectly. Pulse was of natural quickness, small, hard, incompressible. On the evening of December 3rd, there commenced again a series of convulsive fits, which continued until the following evening. On the morning of the 5th vision was very imperfect; a light held before the eyes could be seen with the left eye, but only a glimmer was visible with the right. The ophthalmoscope showed the existence of retinitis apoplectica in both eyes. The patient now continued in a state of perpetual somnolence; but on the night of the 18th she was seized with a new series of eclamptic fits, which were preceded by vomiting, and succeeded by excessive prostration. The disorders of vision still varied in their intensity. On the 11th of January a violent bronchial catarrh with pneumonia brought life to a close.

The autopsy disclosed the following :—

Brain highly anæmic, otherwise natural; dropsical effusions in the serous cavities. *Heart* greatly hypertrophied. *Left lung* air-less in its upper lobe, œdematous in its lower. *Right lung* consolidated posteriorly.

The kidneys were in a state of fatty degeneration with beginning granular atrophy.

The urine was carefully collected and examined while the patient was under observation. It was throughout copious in quantity, even during the severest uræmic attacks; the specific gravity varied from 1010 to 1014; the urea, though always considerably below the normal average, did not sink any lower during the paroxysms.

As a rule, both the quantity of urine and the excretion of urea diminish notably at the period immediately preceding a uræmic attack. Sometimes, however, very great scantiness of urine, or even total suppression (in acute Bright's disease) may exist without evoking any uræmic symptoms. In a case of scarlatinal dropsy related by Biermer, complete suppression of urine continued for 5 days without uræmia; then followed a further period of 4½ days in which urine was secreted, but only in the scantiest proportions (a few tea-spoonfuls a day), and yet no uræmia. At the end of this second period, the urine began to flow abundantly for a short time, and then again became scanty. Three days later uræmic coma set-in, followed by convulsions, which proved fatal.

Uræmic *vomiting and diarrhœa* are common phenomena of Bright's disease. The vomiting which occurs in that disease is not, of course, always uræmic. The digestive functions are notably impaired throughout the complaint, and a heavy or indigestible meal may at any time be rejected as in dyspeptic states from other causes. When the vomiting is really uræmic, it takes place without reference to the nature of the contents of the stomach, and is oft repeated or incontrollable ; the vomited matter is a watery fluid, either distinctly ammoniacal to the smell, or (if acid) evolving ammonia freely when caustic potash is added thereto. The alvine dejections are similarly characterized when due to the same cause.

Paroxysms of dyspnœa belong to the least frequent forms of uræmic disturbance—if indeed such attacks have at any time a genuine claim to the designation uræmic. Fournier cites some cases of this kind. Only the following somewhat doubtful example has fallen under my observation. The case is further remarkable on account of a transitorily ammoniacal state of the urine.

W. R. S., a railway porter, æt. 58, was admitted into the Manchester Infirmary, Dec. 6th, 1860. He was a stoutly-made man, who had led an intemperate life. He had been remarkably healthy, and had scarcely ever lost a day's work. Two months before admission his legs began to swell and then his face. He continued to follow his employment, though with difficulty, until two days before his admission.

On admission, there was general anasarca of moderate degree ; pallid features ; enlarged heart ; copious urine, scantily albuminous, with an abundant deposit of granular and transparent casts, and renal epithelium. Neither casts nor epithelium showed any signs of fatty degeneration. The urine was sometimes highly acid and deposited urates ; at other times it was highly ammoniacal when voided.

On Dec. 27th there occurred a sudden and most intense paroxysm of difficulty of breathing, which threatened suffocation. It resembled in every respect a paroxysm of spasmodic asthma, and lasted five hours. It then passed away, and did not return again with the same intensity ; though slighter attacks of a similar nature occurred on two other occasions.

On Jan. 11, 1861, repeated vomiting took place : there was also a severe cough with a watery expectoration and increasing weakness. Somnolence then set-in, which gradually passed into coma and proved fatal in three days.

The *autopsy* showed hypertrophied *left ventricle;* thickened mitral valve; abundant loose vegetations on the aortic valves. The right auricle and ventricle were filled with a firm voluminous yellow fibrinous clot. With the exception of dense œdema of the inferior lobe of the left lungs the *respiratory* organs presented nothing abnormal. The *kidneys* were found granular with commencing atrophy. The lower urinary passages were quite free from disease.

The ammoniacal state of the urine persisted in this patient for several successive days; and although the secretion was so charged with carbonate of ammonia that it had a pungent smell, and effervesced freely with acids, when quite fresh, the patient experienced no pain or uneasiness about the bladder nor during the act of micturition—which was not unduly frequent. The urine contained no pus. The ammonia in this case must have been derived directly from the blood, and not produced, as is usual in ammoniacal urines, by transformation of urea in the lower urinary passages. Such an occurrence betokened a free generation of ammonia in the blood; there were no uræmic symptoms on the days when the urine was ammoniacal. Did the elimination of ammonia by the kidneys stave off uræmic accidents?

Theories of uræmia.—The absence of anatomical lesions in the brains of persons who die of uræmic coma and convulsions has constrained pathologists to look elsewhere for their determining cause; and by general agreement it has been assumed that that cause consists in certain alterations in the composition of the blood, from the accumulation in it of the excrementitious matters which, in the healthy state, are removed out of the body by the kidneys. The blood thus poisoned is no longer capable of ministering to the tranquil and healthy operations of the nervo-muscular system, and engenders the various abnormities of motion and sense which have been just described. The doctrine of uræmia or urinæmia, thus broadly stated, rests on the following authenticated facts:—(1). Suppression of urine from any cause, is followed, sooner or later, by insensibility and convulsions; animals whose renal arteries or ureters are tied, or whose kidneys are removed, perish with similar symptoms; (2). Certain excrementitious substances have been found in great excess in the blood of persons suffering from uræmic symptoms, namely, water, urea, creatine, creatinine, and the extractives.

But pathologists have not been content with this general appreciation of the matter, and have striven to trace the phenomena to the presence of some one, or the derivatives of some one, of these excrementitious substances in the blood. It will not be necessary in a practical work like the present to enter fully into the controversies—still undecided—which have arisen on this subject. It will suffice to indicate the different views which have been enunciated; and to express my own conviction, after a careful review of the observations and experiments adduced on all hands, that none of the exclusive theories of uræmia have made good their claim to acceptance.

Hammond and Richardson, following the original notion of Willis, contend that the special poison in these cases is urea.*

Frerichs maintains, on the other hand, that urea is itself innocuous; that it may be injected into the veins of animals without detriment; that the mischief in uræmia arises from the transformation of the urea accumulated in the blood into carbonate of ammonia, and that the carbonate of ammonia so generated is the immediate excitant of the nervous symptoms. Frerichs upholds this doctrine by two propositions which he claims to have proved: namely, (1), that carbonate of ammonia invariably exists in the blood of uræmic patients, and in that of animals rendered uræmic by removing their kidneys, and can even be discovered in their expired air; (2), That carbonate of ammonia injected into the veins of healthy animals produces fits of convulsions with intervening pauses of coma, exactly resembling genuine uræmic attacks.

Treitz suggested a modification of this view. According to him urea is not transformed in the blood-vessels, but is first vicariously excreted into the alimentary canal; here it is speedily converted by the gastro-intestinal mucus into carbonate of ammonia; the carbonate of ammonia so formed is then absorbed into the blood, and produces its poisonous effects. That urea is excreted by the intestines in Bright's disease is undoubted, and its swift conversion into carbonate of ammonia has been proved experimentally by Bernard. This theory of Treitz furnishes at least a rational explanation of

* Richter found that solutions of urea (30 per cent.) applied directly to the sciatic nerves of frogs, produced only slight and uncertain convulsions of the muscles, far inferior to those produced by a solution of common salt. A saturated solution of urea produced no convulsions at all.—(F. Richter—Inaug. Diss.: Erlangen, 1860).

uræmic vomiting and diarrhœa, and of the presence of the volatile alkali in the matters so discharged.

Since the theory of Frerichs was first promulgated, however, it has been ascertained by Richardson and Hammond that ammonia naturally exists in the blood of healthy animals: and all subsequent observers (with the sole exception of Petroff) have failed to discover in the blood of animals rendered uræmic by the removal of their kidneys any larger amount of ammonia than exists in the healthy state. It has been likewise shown that other substances than carbonate of ammonia (chloride of sodium, urine, and urea) are capable, when injected into the blood, of evoking comatose and convulsive phenomena.

The recent experiments of Oppler, Schottin, Perls, and Zalesky, seem to have given the *coup de grâce* both to the ammonia and to the urea theories of uræmia; and they indicate, in a very clear manner, that uræmic manifestations depend mainly and essentially on the accumulation in the blood and tissues of those primary products of tissue-metamorphosis (creatine, creatinine, and other extractives), which, in a later stage of histolysis, are converted into urea and uric acid.

But these experiments are still more remarkable for their general bearing on the current views of the functions of the kidneys. It has been hitherto supposed that the office of the kidneys was merely to separate from the channels of the circulation and transmit to the urine, the urea and uric acid which already existed pre-formed in the blood: but it now appears that *urea and uric acid are actually produced in the kidneys;* and that any traces of them found in the blood are due to re-absorption from the urinary channels.

The observations on which these unexpected conclusions are based, are, briefly, the following:—

Oppler[*] found that urea was much more abundant in the blood of animals whose ureters had been tied, than in the blood of those which had been deprived of their kidneys (nephrotomised).

The experiments of Perls[†] on rabbits showed that no accumulation of urea took place in the tissues of nephrotomised animals; whereas in animals whose ureters had been ligatured, urea accumulated rapidly, and was most abundant in 24—48 hours after the operation.

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[*] Beitr. z. Lehre v. d. Urämie. Arch. f. path. Anat. Bd. xxi. p. 260.

[†] Beitr. z. Lehre v. d. Urämie. Konigsberg. Med. Jahrb. Bd. iv. p. 56.

The observations of Zalesky* on dogs, birds, and serpents are yet more remarkable.

In dogs, he found that:—1. The quantity of urea in the blood of healthy and of nephrotomised dogs was almost the same; and that, therefore, removal of the kidneys had no influence in increasing the urea. 2. After ligature of the ureters, urea was always found considerably increased in the blood, the lymph, in the contents of the stomach and intestines, and especially in the tissues. 3. Accumulation of a large quantity of urea in the blood did not increase the ammonia in the blood. 4. Creatine was always considerably increased in the muscles after nephrotomy.

In birds, nephrotomy was found to be an impracticable operation; but ligature of the ureters proved an easy proceeding. The experiments on birds (fowls and geese) yielded the following results:—1. In healthy birds no traces of uric acid or urea could be discovered in the blood. 2. Ligature of the ureters caused death from uræmic coma in 20—37 hours: this operation was always followed by most abundant deposits of urates in the fluids and solid tissues of the body. These deposits showed themselves not earlier than twelve hours after the operation, and were the more abundant the longer the animal lived after the operation. The deposits first appeared in the lymphatic system, then in the blood and all the other tissues. 3. The deposits first appeared in and about the kidneys, and then spread from these as a centre. The lymphatic vessels were gorged with white, amorphous or crystalline, urates, and the serous membranes of the abdomen and thorax thickly encrusted with the same. 4. No increase of the ammonia in the blood could be detected.

In serpents, both nephrotomy and ligature of the ureters were found to be easy operations. The following results were obtained:—1. The blood and tissues of serpents contain no traces of uric acid in the normal state. 2. Nephrotomised serpents lived 18—24 days; ligature of the ureters caused death in about 29 days. 3. In nephrotomised serpents no uric acid or urates was found after death in any part of the body, except a small quantity in the cloaca, where, presumably, it existed before the operation. 4. After death from ligature of the ureters, all the organs and tissues were found strongly infiltrated with urates, which formed thick white crusts and patches on all the mucous and serous membranes, in the joints, in and upon the kidneys, liver, heart, and spleen. By chemical analysis uric acid was also abundantly found in the lungs, muscles, and throughout the body.

Dr. O. Rees believes that the tenuity of the blood in Bright's disease is not without influence in the production of the cerebral symptoms. Traube has still further developed this idea. He contends that the watery state of the blood predisposes to interstitial transudations; that the hypertrophy of the left ventricle increases enormously the lateral pressure in the arterial system; that when, from any cause, a still further increase in the tenuity of the blood-serum occurs, serous transudation takes place through the cerebral capillaries, and gives rise to œdema of the brain. This œdema causes compression of the minute cerebral vessels, and determines an anæmic state of brain, and thereby uræmic convulsions and coma. He is further of opinion that the symptoms are of a comatose character when the œdema and anæmia affect the hemispheres, and convulsive when the central ganglia are the parts affected.

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* Untersuch. ü. d. urämischen Process, etc. Tübingen, 1865.

None of these theories, considered exclusively, explains satisfactorily the protean phenomena of uræmic intoxication, as witnessed at the bed-side. The subjects of Bright's disease suffer under a deep abnormality in the composition of the blood and tissues. The blood is unnaturally watery and poor in albumen ; the blood and tissues are unnaturally charged with the primary histolytic products (creatine, extractives, &c.), and with excrementitious urinary compounds (urea and uric acid), perhaps also with the products of the decomposition of some of these. This state appears to induce in the nervous centres a proneness to sudden disorder and loss of equilibrium. A crisis may at any moment be brought about by an exaltation of one or several of the disturbing elements, or by a supervention of some new and different cause of irritation (hysteria menstruation). A similar hyper-sensitive state of the nervous system prevails naturally in early life ; and an irritation which would be of no moment in an adult (teething, worms, embarrassed digestion, cutaneous irritation, &c.,) suffices, in an infant, to awaken convulsive and comatose phenomena closely resembling those of uræmia.

DIAGNOSIS AND PROGNOSIS.

Diagnosis.—Under ordinary circumstances, chronic Bright's disease presents symptoms, and a condition of urine, so characteristic that it can scarcely be confounded with any other malady. Even when dropsy is absent, a persistently albuminous state of the urine, apart from heart disease, hardly belongs to any other condition.

Temporary albuminuria, as we have seen (see Congestion of the Kidneys), occurs occasionally under a variety of inflammatory and febrile conditions, without structural changes of any importance in the kidneys. These cases differ from Bright's disease in the absence of dropsical effusion; the quantity of albumen is also generally very small; the excretion of urea is natural or even excessive instead of being diminished. When defervescence occurs, it is speedily followed by the total disappearance of albumen.

The real diagnostic difficulties lie:—(*a*) in distinguishing acute and curable cases from chronic confirmed ones; (*b*) in determining the precise anatomical changes going on in the kidneys; and (*c*) in detecting the disease when it is encountered masked by an inflammatory complication or a uræmic paroxysm.

(*a*) The case must be considered as belonging to the chronic and confirmed class, if the disease have crept on insidiously, or if it be found complicated with chronic phthisis, caries, long-continued suppurations, constitutional syphilis, enlarged liver or spleen, or hypertrophy of the left ventricle.

If the invasion have been acute, and the albuminuria still linger after the abatement of the febrile symptoms, time becomes a necessary element in the diagnosis. With every day that passes by without diminution of albumen in the urine, the fear grows stronger that the disease has become confirmed. In such a conjuncture the character of the urinary deposit supplies important information. If the epithelial elements and blood corpuscles continue to be freely discharged, and no, or only trifling, signs of fatty changes appear in the renal derivatives, there is good reason for confidence that the observer has to do with the declining stages of an acute disorder. If, on the other hand, albumen persist in considerable quantity after the pyrexia has passed away, and after blood has ceased, or almost ceased, to appear in the urine, it is probable that the disease has lapsed into a chronic and confirmed state; and if, in addition to these untoward signs, the deposit exhibit marked fatty change, that probability becomes a certainty.

It must not be forgotten that patients suffering from chronic Bright's disease are subject to occasional febrile exacerbations, in which the urine becomes scanty, high coloured, and perhaps bloody. Such exacerbations are liable to be confounded with the acute disorder; and when there are no clear indications of chronicity in the previous history, in the character of the renal derivatives, or in the co-existence of complications, the differential diagnosis of the two conditions may be quite impracticable until the lapse of time shall have cleared up the ambiguity.

(*b*) For the differential diagnosis of the different types of degeneration going on in the kidneys, the reader is referred to the synopses of distinctive symptoms furnished in the first section of the present chapter (pp. 318, 321, 326).

(*c*) When the case comes under notice for the first time, masked by an inflammatory complication (pneumonia, endo-, or peri-carditis, &c.,) a clue to the primary disorder must be sought in the previous history of the case, and in the associated symptoms. When dropsy (or the history of any) is absent, the primary renal disease is apt to be overlooked, and the case regarded as one of simple inflammation of the organ affected: the urine (in such cases) assumes a febrile character, urea becomes abundant in it, and its specific gravity rules high. If, under such circumstances, the quantity of albumen in the urine be but small, the absence of Bright's disease may be counted on; but the converse deduction is not invariably warranted. In pneumonia I have seen the urine for some days "highly" albuminous without, as the sequel showed, the existence of any renal degeneration. In pleurisy and pneumonia (and especially the former) the simultaneous implication of the two sides furnishes a strong presumption (supposing the urine be albuminous) that the inflammation is not simple, but secondary to renal disease. The existence of cardiac hypertrophy without valvular disease, or of notable anæmia, also favours the supposition of Bright's disease.

The differential diagnosis of uræmic coma and convulsions has been already pointed out (see p. 351).

The absence of casts in an albuminous urine gives no security against the existence of renal degeneration; indeed, this absence is generally more apparent than real. When the casts are few in number, and small, they subside very imperfectly, and are apt to escape detection, even with the most careful examination.* In other cases the absence of casts is only temporary; and I have known it most absolute in some of those sad hopeless cases, where the renal disease is the ultimate upshot of an intractable strumous or syphilitic cachexia.

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* About two years ago I was consulted by a medical man who was suffering rom persistent albuminuria. He himself, and another practitioner who was well accustomed to such inquiries, had failed, after repeated examinations, to

Prognosis.—The prospects of a patient suffering from confirmed chronic Bright's disease are exceedingly gloomy. The textural changes in the kidneys are of a kind that do not admit of reparation. The Malpighian bodies become enveloped in an exudation of low plastic material, of which the only tendency is to progressive contraction, and the tubuli are either blocked up with fibrinous plugs or shrivelled into useless fibres. The gland is not, however, equally affected throughout all its parts, and the less injured portions carry on, imperfectly, the depurative functions. As the sounder portions become more and more involved—and there is an almost inevitable, though slow, tendency to this—the work done by the kidneys grows less and less, and the blood is more and more contaminated with histolytic and urinous elements, until at length a limit is approached, which is incompatible with life. Long before this extreme limit is reached, however, death is brought about in a large number of cases by one or other of the numerous complications to which the subjects of renal degeneration are obnoxious.

In certain more favourable cases the structural changes cease to advance, the dropsical effusions (if any existed) are absorbed, and the condition of the patient remains stationary, perhaps for months, perhaps for years; and he may be able, with proper precautions, to prolong existence in fair comfort, and even to pursue light avocations, for very considerable periods of time. Cases protracted to five and six years are not uncommon, and a few instances are recorded in which the patient has survived for ten, fifteen, and even twenty years. The tenure of life under these circumstances is exceedingly precarious, and an imprudent indulgence or exposure may bring life, in a few hours or days, to the verge of destruction;

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detect a single cast in the urine. The specimen of urine sent to me was set aside for twelve hours in an appropriate urine-glass. At the end of that period I could not, after long searching, discover a single cast. Next day, however, the urine deposited an abundant sediment of very minute uric acid crystals. On again searching for casts I found several without difficulty—the precipitation of the uric acid had carried them down.

the patient walks, as it were, on a slumbering volcano, which may at any moment waken its fires with a fatal explosion.

But although the final prognosis in chronic and confirmed cases is thus unfavourable, the immediate prospects of the patient may be fair, and there is still hope that by judicious management amelioration of the more distressing symptoms may be brought about. Except in the ultimate stages of the disease, the dyspeptic symptoms, the irregularities of the bowels, the dropsical accumulations, and the bronchial catarrh, may be combated with good probability of success. The following example is a striking illustration how near an apparent cure the subsidence of the symptoms may proceed, even from an apparently desperate extremity.

Mr. B., a designer, of sober habits, æt. 38, consulted me, May 9th, 1862. He was suffering from great and general anasarca with ascites. The urine was scanty and intensely albuminous. There was an abundant deposit of tube-casts and renal epithelium. These structures exhibited the appearances of advanced fatty degeneration.

The patient stated that the dropsical symptoms had existed a twelvemonth, and had come on gradually—first in the feet, then in the face—without known cause.

A fortnight after, I was requested to see Mr. B. at his own house. He was then confined to bed; the swellings had considerably increased; the legs were tense, and incapable of being moved from excessive œdema; the peritoneal effusion was very great. There was severe dyspnœa (orthopnœa) and frequent vomiting. The urine was almost suppressed.

Taking into consideration the state of the urine, the character of the deposit, and the time the disease had already existed, together with the threatening gravity of the general symptoms, it seemed hardly possible that the patient could rally to anything like seeming health: and yet this took place. Compound jalap powders were administered freely, and blanket baths applied daily. Copious watery motions were produced; the legs burst, and an immense quantity of fluid drained away. Improvement went on steadily in the course of the ensuing month; in September the dropsy and ascites had nearly disappeared. A specimen of urine (of which the flow was copious) was sent to me at this time for examination. It was only slightly albuminous, and after a diligent search I failed to detect any casts.

I did not see the patient after this; but Mr. Briggs, with whom I saw the case, informs me that shortly after, the patient went to Wales; where he continued to improve: the œdema disappeared almost entirely; the appetite returned; and the strength was so far restored that he was able to walk fifteen miles in a day.

On his return to town, however, the dropsical symptoms re-appeared, and increased; and a cough set-in, accompanied with purulent expectoration. The pulmonary symptoms gradually advanced, and he died in September, 1863, about eighteen months after I had first seen him.

The favourable and unfavourable signs in Bright's disease have relation to the state of the skin, the duration of the disease, the degree of deviation of the urine from its natural quantity and composition, and the existence of complications.

The signs which indicate that an unfavourable termination is not far distant are:—obstinate dryness of the skin, the urine, which had previously been abundant, becoming steadily scantier without proportionate increase in the specific gravity, evidence that the disease has existed some years, repeated recurrence of uræmic phenomena, excessive serous effusion, excessive cardiac hypertrophy, a persistently feverish state. Speedy death is indicated by the breaking forth of pneumonia or pericarditis, by suppression of urine or uncontrollable vomiting and diarrhœa. The absence of these signs may be construed in a favourable sense, as indicating a stationary state, and the probability that the final issue may be yet far distant.

An excessive proportion of albumen in the urine, although a proof of the activity of the morbid process, and therefore a sign of evil augury, is not necessarily prophetic of impending death. In a case which I saw with Mr. Stephens, the urine, which was examined almost daily, became constantly solid on boiling, for a period of more than two months. During this period the patient's condition was stationary; he was then seized with fatal pneumonia, of which he speedily perished.

TREATMENT.

In the management of cases of confirmed Bright's disease, three objects are to be especially aimed at, namely: (*a*) to hinder the further extension of the structural changes in the kidneys; (*b*) to prevent the occurrence of uræmic and inflammatory accidents; and (*c*) to palliate or remove certain threatening or burdensome symptoms — anæmia, dropsy, dyspeptic and uræmic phenomena, &c.

To fulfil the first indication, the conditions under which the complaint originated must be carefully traced out, and the patient removed as completely as possible from their further influence. In some instances this is practicable; as when the disease follows intemperance or long continued exposure to wet and cold. In protracted suppurations, necrosis, caries, joint disease, stricture of the urethra and old vesical inflammation, the possibility of the development of renal degeneration should be kept in view by the surgeon, and should have weight in considering the propriety of operation. In all such affections the condition of the urine should be narrowly watched; and the first appearance of albumen is a warning that the opportunity for operative procedures is slipping away, never to return.

There is no evidence that local counterirritants of the severer class—issues, setons, moxas, &c.—applied over the kidneys, exert any good effect; and the ulcerations they sometimes leave are apt to prove intractable. Mustard poultices and tincture of iodine may be applied when the loins are the seat of aching pain; but their influence on the renal lesion is probably *nil*. Blisters are inadmissible on account of their specific irritating effects on the urinary system.

The patient should be habitually clothed in flannel, both limbs and trunk; and the activity of the skin should be further encouraged by moderate walking or carriage exercise, and the occasional use of warm baths and frictions of the surface. The bowels should be opened at least once daily, and the diet should be light and nutritious. Milk agrees well with the majority of this class of patients, and may be freely partaken of. Two or three glasses of claret or hock daily, or a glass of sound beer, are permissible; but the stronger wines and all spirits agree, as a rule, badly, and should not be allowed unless special circumstances imperatively call for their administration.

Medicinal agents of roborant character should be exhibited from time to time—but especially preparations of iron. I have been in the habit, when the secondary symptoms or complications do not call for special treatment, of contenting myself with giving 15 or 25 drops of the muriated tincture of iron in a wine-glass of water night and morning—combined, in cases of strumous affinities, with cod-liver oil. If the tincture produces headache or disturbs digestion, some other chalybeate must be substituted—the citrate of iron, citrate of iron and quinine, the syrups of the phosphate or the iodide of iron, the saccharated carbonate or the ferrum redactum. One or other of these preparations can generally be made to agree. It is important to get patients with chronic Bright's disease to take iron, for saturation of the system with iron is the best safeguard against the profound anæmia which is a fertile source of danger and distress to the sufferers from chronic renal degeneration.

Are there any medicinal substances capable of exercising control over the quantity of albumen lost by the urine? Exact observations do not give an affirmative answer to this question, though a certain reputation has been gained by the mineral acids (especially nitric acid), iodide of potassium, tannin and gallic acid. Dr. Parkes exhibited large doses of tannin and gallic acid without producing any diminution of albumen. I have in a number of cases used gallic acid for a period of many weeks, but could not convince myself in a single instance that it had any favourable influence on the excretion of albumen, and in some instances it occasioned serious gastric disturbance. Oppolzer has recommended alum, and the trials of Heller support this recommendation.

Knowing as we do that persons with albuminuria and degenerated kidneys may preserve passable health for years, so long as digestion is good, the blood not too impoverished, and the complications kept away, the practitioner is not justified in interfering too actively when this stationary condition is maintained; he should confine himself to the enforcement of sound hygienic rules and preventive measures. The patient should be made clearly to understand that he is to treat himself as a valetudinarian; and that in his clothing, his eating, drinking, exercise, and general mode of life, he must go by rule, as the sole condition of not running the most fatal risks.

The most effective means of combating the *dropsical effusions* are hydragogue cathartics and warm baths. For general use there is no hydragogue superior to the compound jalap powder with an additional quantity of the bitartrate. It acts quickly, and procures two or three copious watery stools. The objection to its use is the nausea and sickness which it too often occasions. To diminish this inconvenience as much as possible, an active dose (for an adult ʒiii of the bitartrate, and 15 or 20 grains of jalap corrected with a little ginger) should be administered early in the morning twice or thrice a week. The operation of the medicine passes over in a few hours; and the patient has leisure to recruit himself in the intervals between the doses. This proceeding is less harassing than to keep up a continuous purgation of less activity.

Christison speaks in high terms of gamboge, which he employed in doses of 5 grains, sometimes 7, and very rarely 9 grains, every second day, or in urgent circumstances every day. To prevent griping he had it finely pulverized with half a drachm of the bitartrate of potash. Colocynth, scammony, and elaterium have also been employed on diverse hands. When the serous accumulation is very threatening, and immediate effects are demanded, no remedy is superior to elaterium. It may be given in doses of one-sixth, or one-fourth, of a grain every three or four hours, until free evacuations have been obtained. In the employment of purgatives, it must not be overlooked that exhausting diarrhœa sometimes occurs spontaneously in the later periods of the disease, and that the use of drastics has been known to originate this untoward symptom. It is necessary, therefore, to watch the action of these evacuants, and to immediately desist from their use if the diarrhœa show signs of proving intractable.

Warm baths are unquestionably the most effective of diaphoretics; they not only promote cutaneous transpiration, but often increase the secretion of urine at the same time. They may be applied in all their varied modifications—warm water, hot air, steam, or the blanket-bath. When one modification fails another may succeed. Dr. Liebermeister describes as highly effective, a method of applying the warm water bath, by which the temperature is gradually raised after the bather is immersed. When the patient first enters the bath the temperature is 98°; it is then gradually raised by the admission of warm water to 108°; after remaining in the bath about thirty-five minutes the patient is packed in hot blankets.*

Unpleasant consequences—headache, suffusion of the face, unwonted heat of skin—occasionally follow the use of warm baths, and may even necessitate their abandonment. Generally these inconveniences diminish as the remedy is repeated; and after a few trials patients with chronic renal disease nearly always take their baths with pleasure as well as with advantage.

Of pharmaceutical diaphoretics, Dover's powder, James' powder and liq. Ammon. Acetatis have been chiefly used; their effect is very uncertain.

Diuretics are of much inferior value to purgatives; but when there exists a tendency to spontaneous diarrhœa or to severe gastric derangements, we are constrained to abandon the latter for the former. My experience of diuretics has not given me a high opinion of their efficacy. The testimony of authors on their utility is conflicting. In judging of their effects, some observers have not sufficiently considered that a spontaneous diuresis is the normal outgoing of acute renal dropsy tending to recovery; and that in patients with contracting kidneys profuse diuresis is an ordinary feature of the quiescent state in the middle periods of the disorder, so that when the urine becomes scanty and the dropsical effusions increase during an intercurrent febrile exacerbation, the re-establishment of the diuresis and the diminution of the anasarca on regaining the quiescent condition, must not be too hastily attributed to the diuretic which chanced to be employed pending the pyrexial attack.

Diuretics of the most opposite classes have been recommended by different writers. Bright, who had but slight confidence in diuretics, was in the habit of prescribing uva ursi and pyrola umbellata. Christison relied on digitalis combined with cream of tartar. Rayer perceived little advantage in digitalis or squills, and he found that they almost always, at length, deranged the stomach. Horse-radish tea,† according to his experience, offered of all diuretics the best chance of success. Spruce beer is a much more agreeable beverage, and its diuretic action is probably not inferior. My colleage, Dr. Eason Wilkinson, has repeatedly obtained good results from its use ; and on his recommendation I have tried it myself in a number of cases, with favourable effects ; it agrees well with the stomach, and quenches the thirst, which not unfrequently torments patients with Bright's disease, more effectually than any beverage I know.

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* Prag. Vierteljahrschr., 1861.

† Half an ounce of the root infused in two pints of water was the dose with which Rayer usually began; this was gradually increased to one and one

Tincture of cantharides was employed by Dr. Wells in seven cases, in doses of 30, 50, and even 60 drops per day, with good effect in five. Rayer also reports well of it in some cases ; but he adds, not without reason, "it is an uncertain remedy, which might be dangerous in inexperienced hands."

I have tried in my own practice dandelion, broom-tops, and belladonna with unsatisfactory results.

When other means of evacuating the dropsical effusions fail, and the tension of the integuments threatens erythema and gangrene, there is yet a resource in acupuncture or incision of the legs. This rapid and easy method has the disadvantage that, unless stringent precautions are taken, the punctures are liable to become the focus of erysipelatous inflammation, which may spread and pass into sphacelus, with disastrous consequences. This mishap is quite as likely to follow needle punctures as incisions, and after trial of both plans, I prefer the latter. One or two cuts with a lancet should be made lengthwise in the calf of the leg, or one of them may be placed on the dorsum of the foot. The incisions should be three-quarters of an inch long, and penetrate fairly into the subcutaneous tissue. To prevent erysipelas the following directions should be carried out : the incised member should be wrapped in hot, moist flannels ; these should be changed frequently—at first every two or three hours. At every change, the legs, and especially the incised parts, should be thoroughly sponged with warm water, and the flannels which are soaked with the discharge should be completely cleansed before they are re-applied. Traube recommends that the incisions be frequently

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and a-half ounces. The dried root makes a less acrid infusion than the fresh root, of which a smaller quantity must be used (Mal. d. Reins ii., p. 152).

washed with chlorine water. Under such precautions this treatment may be carried out with safety—always with great relief at the time, and sometimes with prolonged advantage.

In those cases which are characterised by a copious flow of urine (contracting kidney), dropsical effusions, if present at all, are usually slight and partial; and their existence depends chiefly on the watery state of the blood, and the lowered tonicity of the blood-vessels. In these cases, diuretic and cathartic remedies avail little to diminish the œdema: better results are obtained by ferruginous preparations, tonics, and mineral acids. If the patient's general health can by these means be effectually improved, the serous effusions will not delay their disappearance. It is in cases of this class likewise that change of air, or even a sea-voyage, may be recommended, provided always that the disease be not too far advanced.

The treatment of *bronchial catarrh and secondary inflammations* requires to be undertaken with a remembrance of the primary mischief. Mercury and blood-letting are inadmissible—the former (unless in the most guarded way) on account of the peculiar susceptibility of the system in Bright's disease to mercurial preparations, the latter on account of the deep undermining of the strength which has already taken place. Internal antiphlogistics—aconite, digitalis, and antimony—may be freely used; also external applications—chloroform epithems, hot poultices, dry cupping, &c.

The *dyspeptic* symptoms are readily controlled in the early periods by a careful revision of the diet, and the use of vegetable bitters, prussic acid, and antacids. When obstinate vomiting of uræmic origin sets in, it is very difficult to subdue; creasote, morphia, and ice permitted to melt in the mouth, are the most effective remedies. Diarrhœa of similar origin must be combated by acetate of lead, opium, and sulphuric acid.

When *uræmic* symptoms show themselves, renewed efforts should be made to increase the flow of urine, and to awaken the vicarious activity of the skin and intestines by the measures already described. If coma and convulsions have actually seized the patient, further energetic action is demanded. Frerichs, consistently with his view that carbonate of ammonia is the actual poison in these cases, recommends a treatment designed to neutralise the free ammonia, and reduce it to a state of innocuous combination. He directs chlorine (which may be inhaled in the gaseous state or taken dissolved in water) and the vegetable acids to be taken internally, the body to be sponged with vinegar, and vinegar to be used in injections.

During the convulsive paroxysm, chloroform inhalation is the most prompt and ready means of controlling the spasms. When the uræmic paroxysm begins with drowsiness and gradually passes on to insensibility, or when convulsions occur only as breaks in a continuously comatose condition, chloroform affords no prospect of relief.

At the time when Bright, Christison, and Rayer published on this subject, everything in the shape of an apoplectic or convulsive seizure was the signal for immediate and copious venesection; it is not surprising, therefore, to find in the cases they recount that free and repeated bleedings are almost invariably chronicled in the next sentence to that announcing the advent of the uræmic paroxysm. Dr. Richardson has recently advocated the same plan. The immediate effect is, unquestionably, in a large number of cases, to relieve the insensibility; consciousness sometimes returns as the blood flows. But the indiscriminate use of this powerful remedy is the surest way to bring it into ultimate disrepute. A distinction should be drawn according as the renal disorder is acute or chronic, and according to the strength and general condition of the patient. It must be borne in mind that an impoverished and watery state of the blood is an effective factor in the generation of uræmic phenomena, and that a blood-letting, though it may relieve a present attack, increases the predisposition to future attacks. In the uræmic coma of acute Bright's disease, and in certain classes of puerperal eclampsia, the blood is as yet not materially impoverished, and the type of renal mischief is one that gives full hope of eventual recovery, while the attack itself is of extreme danger. In these, venesection—free, and even repeated—is decidedly and urgently demanded. But the matter stands otherwise when the renal mischief is chronic and incurable. The attacks themselves are not so imminently dangerous as when occurring in the acute form of the disease; patients frequently survive repeated uræmic paroxysms without the aid of venesection; the blood is commonly thin and poor; and lastly, there is not any prospect of ultimate recovery. Under these circumstances loss of blood is more likely to shorten than to lengthen life. Further, as Christison remarks (speaking of advanced renal mischief), when the torpor becomes considerable the removal of blood seems of little or no use. In some of the cases reported by Bright, death occurred from coma on the very day of free and repeated venesection. I can only conceive of two contingencies in which withdrawal of blood, in quantity, is justifiable in chronic renal disease; one is, when coma comes on rapidly in a person whose constitution is not, as yet, seriously deteriorated, and whose prospects of life (abstracting the uræmia) may extend to many months or some years; the other is, when there is a necessity for temporary restoration of the faculties paramount to the general chance of prolonging life.

When the comatose symptoms come on gradually, the measures before enumerated should in every case take precedence of blood-letting.

CHAPTER V.

SUPPURATION IN THE KIDNEY: RENAL EMBOLISM.

Howship—A practical treatise on the complaints affecting the secretion and excretion of the urine. Lond. 1823, p. 21.

Rayer—Malad. des Reins. Tom. ii. and iii.

Johnson—Diseases of the kidneys. Lond. 1852.

Todd—Clin. Lects. on urin. dis. Lond. 1852.

Kirkes—Med. Chir. Trans., vol. xxv.

Virchow—Gesammelte Abhandlungen, p. 602.

Traube—Ueber den Zusammenhang von Herz. und Nierenkrankheiten, p. 77.

Beckmann—Archiv f. path. Anat. Bd. xii. p. 59.

Chambers—Decennium pathologicum—kidneys. Brit. and For. Med. Chir. Rev., vol. xxxvi., p. 489.

Purulent formations in the substance of the kidney arise under three conditions, namely: (1), phlegmonoid inflammation ending in circumscribed abscess; (2), multiple abscesses from purulent infection; and (3), occasionally from embolism.*

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* Dr. Johnson describes another form of "suppurative nephritis," in which pus is produced in the uriniferous tubes by transformation of the renal epithelium. This condition he found associated with the occurrence of "purulent casts" in the urine. I have frequently noticed casts of this character, but am disposed to explain their appearance differently. It is not very rare in Bright's disease, to find, in the urine, cells attached to fibrinous casts with double or triple nuclei. But this is no more evidence of pus than the occurrence of the pale corpuscles in the blood (which are, anatomically, indistinguishable from pus corpuscles) is evidence of pus in the blood. When the renal cells proliferate rapidly, they assume very much the appearance of pus corpuscles, and display cleft nuclei.

The cases adduced by Dr. Johnson are quite inconclusive; the first (case 28) was an example of Bright's disease complicated with boils; pyræmia followed, and metastatic abscesses were found in the kidneys and lungs: the second case (No. 29) seems to have been an example of the mottled white kidney acutely

It is necessary to distinguish between abscess situated in the substance of the kidney, and purulent distension of the pelvis and infundibula, with ultimate sacculation of the organ (pyonephrosis). These two conditions were confounded under the common name of "abscess of the kidney," until Rayer pointed out the distinction between them.

1. *Phlegmonoid abscess* is nearly always confined to one kidney. It may be due to external violence (blows or falls on the loins), or to inflammation and suppuration round a calculus or calculi lodged in the substance of the gland, or it may arise as a sequence to suppuration of the lower urinary passages. In the last mentioned cases the abscess is generally due to extension of the inflammation by continuity of tissue along the straight ducts of the pyramids; sometimes, however, where the primary inflammation is confined to the bladder or urethra, the connection of the two (pyæmia being absent) is difficult to explain, except on the obscure idea of sympathy. An abscess formed in any of these ways may involve the whole kidney in destruction, and convert it into a bag of pus.

An abscess of the kidney generally opens into the pelvis of the organ, and its contents are discharged by the ureter. This is by far the most favourable issue. Sometimes the pus works its way out in other directions; it may penetrate the tunica propria posteriorly, and be evacuated in the loins; and recoveries have taken place even after this event, though generally such cases prove ultimately fatal. A renal abscess, more rarely, bursts into the colon or duodenum, and is discharged by stool; or it penetrates into the peritoneal sac, causing rapid death; in still rarer cases, it has been known to push into the cavity of the thorax and be evacuated by coughing.

The symptoms of circumscribed abscess of the kidney are pain in the affected organ, fever, hæmaturia, successive (often regular) rigors; and, if the collection be sufficiently large, a

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developed—complicated with cutaneous erysipelas, but otherwise not unusual in its course. In the third case dropsy and albuminuria of sudden onset had come on in an intemperate and gouty man. Dr. Johnson found purulent casts in the urine, but after a while they disappeared, and the patient survived nearly a year. To apply the term suppurative nephritis to cases like the two last is likely to mislead, for they differ nowise clinically from typical Bright's disease.

fulness or fluctuating tumour is perceived in the renal region. When the abscess bursts into the infundibula there is sudden and copious discharge of pus with the urine—or, if it burst into the intestines, with the stools—followed by simultaneous subsidence of the tumour. In the absence of fulness in the renal region, or of signs of pointing in the loins, the diagnosis is necessarily uncertain. Perinephritic abscess is sometimes accompanied with hæmaturia, and other signs greatly resembling those of abscess in the renal substance.* Albuminuria is by no means a necessary symptom of suppuration in the kidney. It may be wholly absent, or the urine may contain a small quantity of albumen. When abscesses of the kidney form slowly in the course of suppurating disease of the lower urinary passages, they are usually unassociated with any special symptoms, and their existence may not be suspected until the autopsy.

Sometimes the contents of a renal abscess, instead of being evacuated, are gradually inspissated; the liquid parts of the pus are absorbed, and the residue is dried up into a putty-like mass containing shrunken pus corpuscles mixed with considerable quantities of phosphate and carbonate of lime. When an abscess dries up in this manner it may lie permanently latent, and give no further trouble. The destruction of one kidney in this way is compensated by enlargement of the opposite healthy organ, which is thus enabled alone to carry on the renal function.

Treatment.—When external violence has been so inflicted that there is reason to apprehend suppurative inflammation, the loins should be freely cupped or leeched; absolute rest and low diet should be enjoined for several days, and the bowels kept open by emollient glysters. The same treatment should be followed in principle, but modified to suit the accompanying circumstances, if abscess is threatened from renal calculi.

When signs of pointing in the loins are recognised, they should be encouraged by warm poultices; and as the pus approaches the surface it should be evacuated, in order to forestall the danger of evacuation by the more dangerous channels of the peritoneum or thorax. Too often the original cause of the suppuration (impacted calculi or disease of the lower urinary passages) is irremoveable, and the evacuation of one abscess is liable to be followed by the formation of others, which at length exhaust the patient.[*]

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* See a case by Todd. Clin. Lects. on Ur. Dis., p. 39.

2. *Multiple or metastatic abscesses.*—Secondary abscesses are sometimes found in the kidneys, as well as other parts of the body, after death from pyæmia. The kidneys are, however, less frequently the seat of such abscesses than the lungs and liver. In 2161 autopsies performed at St. George's Hospital, Dr. Chambers found pyæmic abscesses in the kidney 12 times: in the lungs 106 times, and 22 times in the liver. The implication of the kidneys has been found in pyæmia from almost every cause—after amputations, lithotomy, lithotrity, and other surgical operations, gangrenous affections, carbuncle, glanders, variola, chronic suppurations, especially of the lower urinary passages. In four instances Dr. Chambers found the kidneys free from abscesses when the pyæmia had arisen from disease of the urinary passages themselves.

The primary disorder (pyæmia) is of such overwhelming gravity, that the renal lesion becomes by comparison unimportant. No symptoms are known whereby the existence of multiple abscesses in the kidneys can be predicated with certainty during life. A strong probability that such abscesses exist, will arise if, with evident pyæmia, the renal regions are painful on pressure and a considerable quantity of albumen be discharged with the urine.

Sometimes the secondary abscesses in pyæmia are almost confined to the kidneys, as in the following example :—

On Jan. 9, 1865, I examined the body of a man, æt. about 45, who had died in the Salford Workhouse, a day or two after his admission, of some obscure disease. The bladder was found thickened and contracted; the mucous membrane softened red and congested but not ulcerated. The ureters were dilated to the thickness of a large quill. The kidneys were enlarged to about twice their natural size, and riddled with hundreds of minute abscesses, the largest of which were about the size of a horse-bean and the smallest like pins' heads. They were distributed through the substance of the organs and on their surfaces, mostly aggregated into groups varying from the size of a sixpence to that of a florin. Each abscess was surrounded with a red inflamed areola. Some of those on the surface appeared thinned away almost to bursting, and in places, resembled a patch of herpetic eruption. Not one of these abscesses had opened into the infundibula; and if pus had made its way into the ureter from the kidney it must have drained along the uriniferous ducts. On section it was seen that the little abscesses displayed a general tendency to range themselves end to end, in lines following the direction of the ducts of the pyramids.

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[*] Illustrative cases of abscess of the kidney may be found (in addition to those indicated at the head of the chapter) in the following sources :—*Lancet*, 1847, i., p. 335; 1853, i., p. 32; 1863, ii., p. 69. *Med. Times and Gaz.*, 1854, i., p. 23; and ii., pp. 241, 343. *Med. Gazette*, vol. xix., p. 888; xxiv., p. 563; xxvii., p. 141; xlv., p. 252. *Path. Soc. Trans.*, 1849—1850, p. 234; vol. v., pp. 178, 179; xiii., p. 131. *Gaz. Hebd.*, 1863, p. 40. *Dublin Hosp. Gaz.*, 1854, p. 147; and vol. i., p. 121. *Med. Chir. Rev.* (1824—1834), new series, xii., p. 81; xix., pp. 159, 234. *Med. Commentaries*, vii., p. 41. *Med. Facts and Obs.*, vi., No. 3. *Encycl. d. Sc. Med.*, v. 54, h. 19. *Wilson, Lects. on dis. of ur. organs.* Lond. 1821, pp. 281, 283. Bennett, Clin. Lects., 2nd ed., p. 731. Ulrich, cited by Rosenstein, l. c., p. 287.

There were about 8 oz. of urine in the bladder: it deposited, on standing, an abundant sediment of pus; it also contained a good deal of albumen—more than the pus accounted for. Careful examination failed to discover casts of tubes of any sort.

Both lungs contained a few secondary abscesses; there was abundant recent pleurisy on both sides. The right heart was somewhat dilated; otherwise the organ was healthy. The liver was healthy.

The pyæmia in this case appeared to have arisen from chronic cystitis.

3. *Renal embolism.*—It was well known to Rayer that rheumatic endocarditis was sometimes attended with the formation of numerous deposits of a yellow colour in the kidneys, which he considered to have the nature of plastic lymph. He described and figured such cases under the designation of "nephrite rhumatismale."* But it was not until the doctrine of embolism was worked out by Kirkes, Virchow, and others, that the real nature of these lesions was understood.

The loose fibrinous vegetations which beset the aortic and mitral valves in endocarditis are apt to be detached, and swept away with the current of blood into the arterial system, and to be finally impacted in some small artery in the brain, kidneys, or other part. But it is not solely in endocarditis that such masses are dislodged and carried away. The same may happen in atheromatous erosions of the valves and aorta; or a portion of the fibrinous lining of an aneurism may be detached and whirled away to the kidneys and other places.†

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* Rayer, l. c., t. ii., p. 73. Atlas, pl. v.

† See the history of a case of aortic aneurism, by Murchison and Moore in vol. xlvii. of the Med. Chir. Trans., p. 129.

The effect of the lodgment of plugs or emboli from any of these sources in the arteries of the kidneys varies with their magnitude. Small emboli either do not produce any appreciable symptoms, or they merely occasion a dull uneasiness in the renal region; and their existence is only ascertained by inspection after death. But if one of the larger arteries is plugged up, not only does the occurrence produce symptoms referrible to the kidneys (sudden acute pain in the loin shooting down the ureter), but it may, under favourable circumstances, even be diagnosticated during life.*

In three cases of embolism described by Kirkes (in which death ensued from softening of the brain, consequent on obstruction of one of the main cerebral arteries, by a fibrinous plug derived from warty vegetations on the valves of the left heart), the kidneys were the seat of numerous masses of yellow deposit surrounded by red areolæ. An injection thrown into the renal artery did not penetrate in the least degree into these yellow patches. The further researches of Virchow and Beckmann have added some particulars to the description of Kirkes. The yellow spots are situated almost exclusively in the cortical substance; they are frequently wedge-shaped, with their bases bulging underneath the tunica propria, and their apices pointing toward the infundibula. They vary in size from a hemp-seed to a hazel-nut. At first they look like red hæmorrhagic patches; in the centre of each there soon appears a yellow spot. This enlarges by cell-growth, and either softens in the centre (breaking down into a fatty *débris*, more rarely into genuine pus) or finally contracts into a cicatricial remnant, with destruction of the adjacent secreting tissue. It is necessarily a matter of extreme difficulty to demonstrate the existence of obstructions or plugs in the minute vessels at or near one of these yellow spots, and some doubt yet hangs over the demonstration. When one of the main branches is obstructed, the embolus is more easily discovered, and the appearances are somewhat different. The first effect of cutting off the arterial supply of a considerable section of the kidney is to produce intense hyperæmia of the surrounding parts, which results in rupture of the Malpighian capillaries and effusion of blood into the surrounding tissue. In this way a wedge-shaped apoplectic area is formed, embracing the whole thickness of the organ.

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* Such a case is related by Traube, l. c., p. 77.

As a rule, the effects of embolism in the kidney are of very slight clinical importance; they pass by in the immense majority of cases, without recognition; their occurrence is always secondary to some much more grave primary disorder, which altogether dominates the prognosis and treatment. Sometimes the larger ones go on to suppuration, and, still more rarely, to gangrene.

CHAPTER VI.

PYELITIS AND PYONEPHROSIS.

Rayer—l. c., t. iii.
Bright—Memoirs on Abdominal tumours (New Syd. Soc.), p. 224.
Howison—Case of Sero-purulent distension of kidney. Edin. Med. Journ., 1822, p. 557.
Basham—On dropsy. London, 1862, p. 265.
Oppolzer—Wiener Med. Wochenschr., 1860.
Todd—Clin. Lects. on dis. of urin. organs. Lond. 1852.
Chuckerbutty—Lancet, 1860, ii.
Mosler—Archiv der Heilkunde, 1863, p. 420.
Kussmaul—Würzb. Med. Zeitsch., 1863, p. 56.
Pathological Soc. Trans., vol. i., p. 117 ; and vol. x., p. 209.

Morbid Anatomy.—Inflammation of the pelvis and calyces of the kidneys, or pyelitis, may be acute or chronic ; it may involve the two sides, or be confined to one.

In *acute* pyelitis the mucous membrane is injected ; sometimes minute echymoses dot its surface, and blood may be effused on it ; the epithelium is more or less freely shed, and at a later period pus is formed. In rare cases the surface is lined with false membranes (diphtheritic pyelitis) which are liable to be detached, and to block up the ureter.

In *chronic* pyelitis the mucous membrane has a dead-white colour, sometimes grey, or slate ; either it is not at all injected, or it is traversed by dilated veins. The membrane is also thickened, and the pelvis and infundibula are dilated. This dilatation, as it proceeds, encroaches more and more on the substance of the gland: first the papillæ are flattened or obliterated; next the pyramids, and finally the cortex are gradually annihilated, and the organ is wholly excavated (or sacculated) and transformed into a multilocular pouch filled with pus. Rayer describes and figures examples in which the pelvic membrane was studded with minute vesicles resembling sudamina.

When the disease is due to the lodgment of calculi, ulceration may exist on the mucous membrane, and sometimes these have been known to lead to perforation, with effusion of pus and urine into the surrounding cellular tissues, or into the intestines or peritoneum. These accidents usually occur after sacculation and dilatation of the kidney have taken place, but sometimes when the organ does not transcend its ordinary dimensions.

When the pus and urine lodged in an excavated kidney fail to find a free exit through the ureter, from blocking up of its channel by a calculus, a clot of blood, thickened pus, tuberculous or cancerous *débris*, &c., these fluids accumulate behind the obstacle, and distend the organ into an abscess-like cavity (pyonephrosis), which sometimes forms a palpable tumour in the flank. The matter so incarcerated may open a way for itself in any direction—backward through the loin; downward along the psoas muscle into the iliac fossa, or under Poupart's ligament; upwards into the bronchial tubes, though this is rare; more frequently it penetrates into the duodenum or colon; or lastly, into the peritoneum.

In the cavity of the inflamed pelvis there are often found, in addition to pus and urine, blood, urinary calculi of various shapes and number, calcareous crusts, hydatids, tubercle, cancer, or whatever other foreign or adventitious matter may have been the cause of the inflammation.

If the urine remain acid, uric acid and the urates may be precipitated in the interior of the pelvis; but if it become ammoniacal, as it commonly does in advanced cases, the mixed phosphates are thrown down. These are sometimes produced in great quantity, and mingling with the purulent contents of the sac, thicken the whole into a semi-fluid mortar-like substance. In other cases, the phosphatic matter forms incrustations, which adhere in places to the walls of the cavity, or lie loose as friable concretions. Sometimes, again, the excavated organ, instead of forming a tumour (or as a subsequent stage to such tumour), slowly contracts, until at length it is reduced to a shrivelled pouch weighing only a few drachms.

In other instances, the pus is gradually inspissated and impregnated with mineral matter (carbonates and phosphates of lime and magnesia) until it is converted into a putty- or chalk-like material, which fills up the compartments of the sacculated kidney. Sometimes the fibrous septa which separate the compartments are extensively calcified. In an example of this kind (exhibited by Dr. John Medd to the Manchester Medical Society) which was handed to me for examination, a saw was

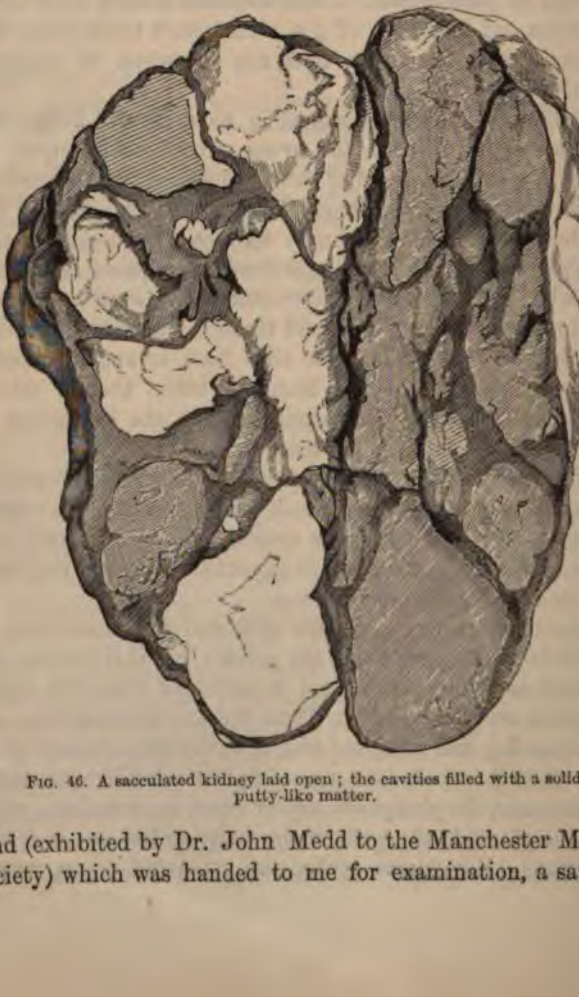

FIG. 46. A sacculated kidney laid open; the cavities filled with a solid putty-like matter.

required to cut the kidney across, and a piece of one of the bony septa which was ground down displayed, under the microscope, the characters of true bone, though in a rudimentary state. A fine specimen of similar transformation is preserved in the museum of the Manchester Infirmary, of which the drawing on the opposite page (Fig. 46) is a representation.

It rarely happens, in cases of chronic pyelitis, that the other parts of the urinary apparatus are free from disease. The most common combination is chronic cystitis with dilated, thickened, suppurating ureters. The substance of the kidney, or what remains of it, is likewise involved at length in a degeneration of the nature of chronic Bright's disease (pyelo-nephritis) ; and if both kidneys are affected, the usual symptoms of that disorder present themselves—general anasarca and characteristic changes in the urine.

Etiology.—The symptoms of pyelitis, and the varied accidents which it may present during its course, whether acute or chronic, have so direct and intimate a connection with the cause of the inflammation, that practically it is necessary to couple the description of the different species of pyelitis with an etiological condition. This is so far true that the designation, pyelitis, expresses nothing more than an anatomical fact. As a nosological heading it includes numbers of cases which have little real clinical affinity. In a certain number of cases the inflammation of the pelvis and its appendages is an important, perhaps the most important, feature of the patient's complaint; but in the majority of cases pyelitis is a subordinate and often insignificant incident in the history of some graver disease. Pyelitis may arise under very varied conditions, viz, :—

(1) From over-doses of turpentine, cantharides, and other stimulating diuretics.

(2) Some degree of pyelitis usually accompanies both acute and chronic Bright's disease and diabetes.

(3) Certain general diseases are sometimes complicated with a degree of pyelitis—typhus and other eruptive fevers, pyæmia, scurvy, diphtheria, cholera, carbuncle, &c.

(4) From mechanical irritation produced by the presence of a foreign body in the pelvis of the kidney or infundibula—

urinary gravel and calculi, hydatids and other parasites, blood-clots, cancerous and tubercular deposits.

(5) From stagnation and decomposition of the urine in the pelvis and infundibula. Simple stagnation of the urine (without decomposition), from an obstruction in the ureter, usually causes only dilatation (hydronephrosis); but if it occur suddenly, the pressure of the dammed-up urine may excite acute pyelitis.* Severer inflammatory changes occur if the stagnant urine becomes decomposed, and its urea converted into carb. ammonia. It is probable that the intractable, generally fatal, pyelitis which sometimes follows pregnancy arises in this way.

(6) From extension upwards of inflammation from the bladder. This is a frequent cause of the worst forms of pyelitis. In whatever manner cystitis may have been engendered—whether by a urinary calculus, enlarged prostate, fungous or tuberculous disease of the bladder, or stricture of the urethra—it can scarcely persist in intensity for a lengthened period without producing some or all of the following consequences :—dilatation and suppuration of the ureters, pelvis, and infundibula, and ultimately sacculation of the kidneys and destruction of the renal tissue.

(7) From cold and unknown causes. It is very rare that pyelitis is not secondary to some antecedent morbid process or mechanical irritation; but now and then cases are met with, in which pyelitis exists without any definite antecedent to account for it, as in the following example :—

In March, 1857, I admitted into the Manchester Infirmary a man greatly emaciated, with hectic symptoms. The urine contained a large quantity of pus; its reaction was acid; it contained no casts of tubes, nor more albumen than the pus accounted for. The patient stated that his water had been milky for more than a year, and that his health had been gradually failing for about the same time. He had never passed any gravel, nor had he ever suffered from nephritic colic. As far as he knew, the urine had never been bloody. He attributed his complaint to the nature of his occupation, which was to manufacture bichromate of potash. He died eleven days after admission. At the autopsy the thoracic organs were found perfectly healthy, as were also the liver, spleen, and intestinal tract. When the bladder was opened, some injection of the mucous membrane was found, but it was not thickened, and the viscus was not contracted.

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* See a case reported by Brunner in the Verhandl. d. phys-med. Gesellsch. in Würzb. viii., p. 146.

Both ureters were dilated to about double their usual size, and filled with pus. The two pelves and the infundibula were enlarged, and their lining membrane opaque, and bathed in pus. The kidneys presented very slight signs of disease; the papillæ were flattened and yellowish, as if they contained pus within their ducts; the remainder of the renal tissue appeared healthy. No foreign body was detected in either pelvis, and the path of the urine was free throughout. Death could only be attributed to the long-continued exhausting purulent discharge, which had been allowed to go on without an attempt to check it until within eleven days of his death.

Symptoms.—The symptoms of pyelitis are compounded of those directly due to the inflamed state of the pelvis and calices, and of those of the primary lesion which is the exciting cause of the inflammation. Only the former will be dealt with in this connection; the latter will be described under their appropriate headings.

An aching pain and sense of weakness in the back are rarely altogether absent in pyelitis. The pain may be confined to one loin or affect both, according as the disease is single or double. Sometimes, however, single pyelitis is accompanied with pain over both kidneys. The pain is increased on pressure.

Symptoms of nephritic colic are generally noted at one time or other, or repeatedly, when the disease is due to the lodgment of a stone. Similar attacks are also common in pyelitis from hydatids; sometimes also in tuberculous and cancerous pyelitis.

The most important *direct* symptoms of pyelitis are found in the altered characters of the urine. In the early stage the urine contains blood (often only in microscopic quantity), mucus and epithelial cells from the pelvis and infundibula. The appearance of these last affords the most certain diagnostic indications. The pelvic and infundibular cells are very irregular, spindle-shaped, tailed, three-cornered, elongated, rudely circular, &c. (see Figs. 19, p. 88, and 43, p. 333). The urine is usually acid. The quantity of albumen in it only corresponds to the admixed blood and pus.

In the more advanced stages, the characteristic epithelium just referred to is usually replaced by pus, which may be discharged in large quantities. The urine is still commonly acid; but as the sacculation of the kidneys proceeds, the mingled pus and urine are liable to decomposition, and the urine becomes ammoniacal.

If the urinary channels remain free, the discharge of pus is constant and regular; but if, as frequently happens, the ureter is blocked up by a calculus, a hydatid, a clot of blood, a mass of viscid pus or other *débris*, the discharge of pus is for a while arrested; and if the disease be confined to one side, the urine temporarily recovers its transparency and healthy characters. When the obstacle gives way, pus suddenly re-appears in great quantity in the urine. If the distension of the pelvis have proceeded to the formation of a tumour in the flank, the intumescence is necessarily greatly influenced by the formation and removal of such an obstacle. When the discharge of pus diminishes, the fulness in the flank increases, and becomes painful; when the course of the pus is re-established, the tumour suddenly subsides, and the urine becomes again loaded with pus. This train of events throws a strong light on the nature of the case. The stoppage in the ureter may persist for varying periods—a few days or a few months—or it may prove permanent. When both sides are affected the obstruction of one ureter diminishes, but does not entirely dissipate the pus from the urine; the same is also the case when the impediment is partial.

Micturition is always more frequent than natural in pyelitis; and during the nephritic attacks, it is painful and incessant.

Rigors are of frequent occurrence, especially when there is tumour; they sometimes assume a quotidian periodicity—recurring every evening with tolerable regularity. Well-marked hectic is often present in the later periods.

The bowels are frequently disordered. Unmanageable diarrhœa usually prevails, induced doubtless by the inflammatory adhesions which take place between the dilated kidney and the colon which passes over it. In other cases (when there is tumour) the bowels are obstinately constipated, and require the frequent use of enemata. This is occasioned by the pressure of the tumour on the colon; in one case, related by Bright, the descending colon was so contracted from the pressure of a pyonephrotic tumour, that it was reduced to the condition and appearance of a thick cord.

The occurrence of *tumour* in the flank is generally a late event in the course of pyelitis. This tumour is usually the seat of fluctuation, often obscure; and is commonly painful and tender on pressure. It is dull on percussion, except where it is crossed by the colon. When the tumour is on the right side it is separated from the liver by the transverse colon; when, however, adhesions form between the sac and the under surface of the liver, this sign may be wanting. The tumour is subject to important variations of size, as already explained, according to the open or obstructed state of the outflow from it into the bladder. In some cases the tumour is so large that it extends across the middle line; more commonly it amounts only to a fulness in the loin or in the space between the crest of the ilium and the false ribs. The outline of the abdomen is thus rendered unsymmetrical.

The ultimate issues of pyonephrosis are diverse; scarcely any two cases run a parallel course. The various directions in which the sac may burst have already been noticed (p. 382), and the symptoms vary correspondingly. But the sac may not burst at all, and the patient dies exhausted by the wasting discharge. This is indeed by far the most common termination. Or again, things may take a more favourable turn; the discharge gradually diminishes, and, finally, ceases altogether; the sac contracts and dries up, and, if the opposite kidney remain sound, perfect restoration to health takes place. Or the restoration may take place differently: the purulent collection, instead of being discharged, dries up into a putty-like mass, and ceases to give further trouble.

The following abstracts of cases will serve to illustrate the course and symptoms of some of the chief types of chronic pyelitis. Other illustrations will be found in the chapters treating of parasites, tubercle, and cancer in the kidneys.

CASE I.—*Double calculous pyelitis.* (*Dance, Archives Gén.* xxix., 149.)

A young woman, æt. 23, was admitted into the Hôtel Dieu, Jan. 12, 1824. She had experienced, two years and a half before, a tedious illness, which commenced with hæmaturia, accompanied by fixed and continued pains in the renal region. Subsequently, the urine became turbid and purulent; it was passed in small quantity and frequently. At the end of eighteen months, after the application of a large number of leeches to the loins, the health improved. The renal pains gave place to a habitual sense of weight in the loins; the urine, however, continued purulent. Three weeks before, the menstrual discharge was suddenly suppressed from cold; and when the patient came under observation the face was drawn, the eyes sunken; there were severe abdominal pains increased by pressure: these were especially severe in the lumbar region. The urine was turbid, scanty, and voided with pain. Leeches were freely applied. But obstinate vomiting came on, and the patient died in five days.

Autopsy.—The kidneys were enlarged to about a third above their ordinary size; their surfaces were nodulated, and unnaturally hard, but presenting here and there points of fluctuation. On cutting open the organs they were found extensively sacculated and full of pus. The left kidney contained nine calculi, and the right fifteen; these were lodged in the dilated calices. The proper substance of the kidney was expanded and attenuated, but otherwise healthy. The ureters were dark coloured, marbled on the surface, and their lining membrane thickened.

CASE II.—*Tumour formed by the left kidney (pyonephrosis), discharging pus copiously both by the urethra and the rectum, depending on a large renal calculus.* (*Bright, loc. cit.*, p. 227.)

A man, æt. 40, first seen by Dr. Bright, April 30th, 1836, had, for the last twenty years, experienced occasional pain in the left side, which he ascribed to a blow; he had, likewise, at times, felt pain in passing urine, which was then turbid with deposit; but about three months only before Dr. B.'s visit, had a tumour been detected or suspected in the left lumbar and iliac regions. He was found considerably emaciated. The urine was neutral, with a very disagreeable smell, and contained a large quantity of pus with a little blood. The whole quantity of pus passed daily was from four to six ounces.

A tumour existed on the left side of the abdomen descending far below the umbilicus, hard to the touch, and fixed in the left lumbar and iliac regions. It felt smooth and even, and was rather tender at one point.

A month later (June 1st) the tumour appeared to occupy nearly the situation of an enlarged spleen, but Dr. B. thought he felt the colon passing over it. The urine passed in twenty-four hours contained only three ounces of pus. The perspirations were profuse.

June 6th.—He had suffered lately a good deal of pain in the left side, and was evidently feverish; he was accordingly directed to leave off the tonic and nourishing medicines and food he had been taking.

June 15th.—Two days after the last visit diarrhœa came on, accompanied with tenesmus. The tumour was found now greatly diminished; and on examining the stools it was evident that a large quantity of pus was passing that way. The discharge of pus with the urine was undiminished, but there could be no doubt that the abscess in the kidney had ulcerated into the descending colon. The patient lived for about six weeks after this; hiccup came on and proved very obstinate; and pus continued to be discharged both from the urethra and the rectum.

Autopsy.—When the abdomen was opened, the left kidney was seen occupying the space from the diaphragm to the brim of the pelvis, and along its whole length passed the descending colon, much contracted. There was a small fistulous opening, not larger than sufficient to admit a goose-quill, from the sac into the sigmoid flexure of the colon as it passed over the lower part of the kidney just at the point where peculiar tenderness had been early observed, and here the intestine looked a little drawn in. The pus was found to have escaped into the psoas and lumbar muscles very extensively. On removing the left kidney, and examining it more accurately, it was found to contain a large coral-formed, lithic-acid calculus, extending its branches into all the cavities of a sacculated pelvis. The kidney was full of pus, and in several parts cerebriform matter was sprouting into the cavities with most luxuriant growth, into which tufts of vessels were seen entering.

Cases of this class, being surrounded with more or less obscurity as to the exact state of things within the abdomen, are more instructive when the revelations of the autopsy are at hand to illuminate the clinical history; but the nature of some of the cases which end in recovery, are so clearly indicated by their symptoms, that they may be cited with advantage, and without any doubt as to their real nature.

The following is from Dr. Todd (*loc. cit.*, case xlviii.) :—

Case III.—A female, æt. 25, unmarried. She had been passing pus with the urine at least a twelvemonth before admission into hospital, and in considerable and constant quantity. For the last five years, she had suffered pain in the loins, referred especially to the region of the right kidney. This pain varied in intensity; it was generally slight and dull, but now and then severe. There had been no symptoms of an acute attack, nor any rigors or vomiting. She never, to her knowledge, voided blood in the urine, nor ever passed any gravel or calculus; nor did she ever seem to have suffered from severe pain in the direction of the ureter.

Rather more than a twelvemonth before her admission into hospital, she was suddenly attacked with retention of urine, which lasted twenty-four hours: and immediately after its cessation, she first began to notice in the urine a sediment, which presented a purulent character. This attack of retention of urine was preceded by slight rigors, but the constitutional disorder was of so mild a character as not to cause her to lie up at all.

When admitted into hospital, pus was passed daily with the urine to the extent of two to four ounces; yet there was but very slight general ailment.

On examination, a very large tumour was found situated in the region of the left kidney, forming a considerable projection beneath the abdominal wall. This tumour, which was three times the ordinary bulk of the kidney, was elastic and yielding to the touch, and communicated the sensation of a soft elastic swelling filled with fluid. There was dulness on percussion all over the surface of the tumour, which was smooth, round, and free from any notches or projections.

The tumour was not tender; the patient could bear it to be handl
without pain, unless hard pressure were used, when she complained of
dull pain. Her most urgent symptom was an occasional cutting pai
referred to the neck of the bladder, sometimes accompanied with slig
difficulty of micturition. She stated that occasionally she had a sensati
of fulness in the left side, which would go off rapidly, as if something h
burst, and then there would very soon follow an increased flow of pus
the urine. Sometimes as much as eight ounces of pus would be passed
the twenty-four hours. Dr. Todd diagnosticated a stricture of the urete
probably near the bladder, causing backward pressure on the kidney, wi
dilatation and sacculation of the organ.

Eighteen months afterwards, the patient presented herself again to D
Todd. She stated that, on quitting the hospital, she went to Brighto
there she improved in health very greatly, and the purulent dischar
gradually diminished. On a careful examination of the side there was
trace of tumour; only a few pus globules could be detected in the urin
and it was doubtful whether these were not derived from the vagina or t
bladder.

CASE IV.—*Pyonephrosis from calculus in the ureter without purule urine.* (*Howison, Ed. Med. J.* 1822, *p.* 557.)

A medical practitioner, æt. 25, had severe and protracted nephrit
symptoms on the left side at the age of 15. He recovered from th
attack, but during the subsequent years he suffered repeated paroxysms
pain in the left kidney, extending to the umbilicus. The urine was
times scanty, and once or twice slightly tinged with blood, but it nev
was observed to be milky, or to contain anything like pus.

Between the paroxysms his health was good; he followed the practi
of his profession, and underwent a good deal of fatigue. The most di
tinguishing symptom in these paroxysms was fixed pain, of a gnawi
description, extending from the spine toward the umbilical region, increase
by pressure, even the slightest, during the severity of the attack. The
was habitual constipation, and a most unusual sensitiveness to cold.

His last attack but one occurred in February, 1821; he recovered fro
this in about six weeks, after being bled to 130 ounces. He became lus
and florid after this attack, and was able to take long journeys in his gi
but riding caused so much pain that he was obliged to give it up.

About September he underwent a good deal of fatigue; and it was su
posed that he suffered a good deal of pain, from being observed repeated
to bend his body forward for relief, although he would not allow it whe
questioned.

One Thursday evening, toward the end of September, he went to b
earlier than usual, complaining of fatigue. He rose next morning at seve
o'clock, and his last fatal attack commenced at eight. The symptom
resembled those of the former paroxysms; there was intense pain in th
left renal region, and a hardness was perceived when the hand was applie
to the seat of pain, with a peculiar sense of crepitation. The symptom
became rapidly aggravated, and, notwithstanding all the means employe

including the abstraction of blood to the extent of 150 ounces (!), he died on the fourth day.*

Autopsy.—The left kidney and pelvis were found converted into a reniform sac, a foot long, and nine inches broad. The surface of this sac was marked out into three lobes. When opened, it was found full of a fluid resembling pus, mixed with serum. The renal substance had wholly disappeared, except a few small portions, leaving nothing but a cavernous cyst, consisting of the proper external membrane of the kidney, and its internal membrane much thickened. It was divided into three large irregular cells, freely communicating with the dilated pelvis, into the apex of which the ureter (of its natural size) opened. The septa between the cells were hard like cartilage with thickened edges.

The orifice of the ureter was closely blocked up by a small calculus.

CASE V.—*Stone in the bladder for* 16 *years:—removal by the recto-vesical operation:—death five years after from pyelitis.*

James H., æt. 21, was admitted, under my care, into the Royal Infirmary, September, 1858, labouring under symptoms of stone in the bladder. On sounding, a large concretion was forthwith detected. The patient stated that he had been subject to difficulty and pain in making water since he was five years of age. When he came under treatment he was emaciated almost to a skeleton, and unable to leave his bed. Micturition was excessively frequent, the urine ammoniacal, and loaded with viscid pus. By rest in bed and anodyne treatment, the symptoms diminished greatly in severity; and on the 17th of December he was judged by my colleague Mr. Southam, who now took charge of the case, to be fit for operation. The stone was removed by the recto-vesical section. It weighed over 4½ ounces, and contained a nucleus of oxalate of lime, overlaid with an immense mass of secondary phosphates.

The patient made a slow recovery from the operation. By the end of April, 1859, the fistulous communication between the bladder and rectum appeared closed; and the patient was discharged in excellent health, rapidly gaining weight.

The subsequent history of the case, up to the time of his death, extends over a period of five years. Soon after leaving the infirmary, the recto-vesical fistula re-opened, and it never afterwards could be completely closed. The general health continued good, and no practical inconvenience arose from the fistula, until about a twelvemonth before his death. The purulent discharge with the urine then began to increase; he lost flesh, and gradually sank in the early part of 1864.

Autopsy.—The bladder, ureters, and kidneys were removed entire. The bladder was contracted and thickened; the ureters were dilated to the size of the little finger, and were long and tortuous; both kidneys were extensively sacculated, but not enlarged, and filled with pus; and the

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* It seems highly probable from the narrative that death was directly caused by loss of blood; the patient insisted on venesection; he tightened the ligature, and *bled himself* on the night of his death.

secreting tissue was reduced to a thin layer of cortical substance scarc
half an inch thick.

CASE VI.—*Pyelitis with tumour, after parturition.* (*Bright's Memo*
on Abd. Tumours, New Syd. Soc. p. 212).

A woman, æt. 30, was admitted into Guy's Hospital, June 13, 1832. S
had a large abdominal tumour. It occupied a situation which extended ov
nearly half the abdomen, not very different from that of a greatly enlarg
spleen, but running back more completely into the lumbar region, a
there affording a tense, somewhat elastic feel. It appeared to be perfect
fixed; even when the patient was turned completely on the right side,
did not shift its place. It felt as if fixed to the ribs themselves, und
their margins, which were obviously protruded a little by its bul
Towards the lower parts, and particularly below the crest of the ilium, a
descending towards the pelvis, the enlargement felt much softer and l
tense. Dr. B. was at once convinced that the tumour depended on a diseas
kidney, and it seemed likely that the softness of the lower part mig
arise from a portion of the intestine, which probably was the colon passi
over the kidney.

Three years before, the patient had suffered for many months from freque
micturition, with pain and forcing—the urine being occasionally tinged wi
blood. Eighteen months after, she was put to bed with a living child, a
about six weeks subsequently, she first discovered the tumour. Since th
however (nine months before her admission), she had borne another livi
child, and about Christmas she began to pass considerable quantities
what she considered "matter" with the urine.

On admission she was feeble, and looking hectic, with frequent calls
pass urine, and pain in doing so. The urine, which was acid, contain
pus. Some days the quantity of pus was very small; but on other da
as much as six or eight ounces of pure pus were collected; and afte
large discharge, the tumour was often decidedly reduced for a day or tw
The bowels were costive. About the 13th of July, chest symptoms set
with diarrhœa, under which she sank.

Autopsy.—The tumour proved to be the distended left kidney reachi
from the diaphragm to the brim of the pelvis. The descending colo
contracted like a thick cord, ran longitudinally on the surface of the tumo
The tumour was adherent to the colon and the lumbar parietes. T
flattened pancreas lay across its surface, in its anterior and inner aspe
The ureter was thickened, and resembled an artery, but its canal was
no means proportionably large. It was traced to the bladder, where
orifice formed a permanent opening, into which a goose-quill could eas
have been inserted, and the membrane was tuberculated. The blad
was exceedingly small; the uterus natural. The tumour contained abo
a pint and a half of healthy, well-formed pus, lodged in cells communic
ing with the pelvis of the kidney, and apparently formed by the distend
infundibula.

The right kidney was healthy, as were also the other abdomin
organs.

Diagnosis.—(*a*). *Pyelitis without tumour.*—In the first stage of the complaint, the presence of the characteristic epithelium of the pelvis and calices in the urinary deposit, generally suffices to indicate the nature of the disease. When the urine has become purulent, these may still be found mixed with the pus corpuscles; but in more advanced cases this valuable sign is no longer available, and the source of the discharged pus must be traced by other indications. These indications are often more of a negative than positive character. When pus is discharged with an acid urine, and signs of disease of the bladder, prostate, and urethra, are absent, the *primâ facie* inference is, that it comes from the pelvis of the kidney:* this inference is strengthened almost to a certainty, if tenderness exist in either loin, or if there be any history of antecedent nephritic colic.

It is much easier to recognise the existence of pyelitis when it stands alone than when it co-exists with, and is perhaps the consequence of, chronic disease of the lower urinary passages. Pyelitis is a common complication of old standing cases of cystitis, enlarged prostate, and urethral stricture. In the absence of tumour in the flank it may be impossible, in such cases, to arrive at a positive certainty as to the co-existence of pyelitis. Little help can be obtained from the character of the urine, because it bears the stronger impress of the vesical prostatic or urethral disorder: but a careful weighing of the following points will generally lead to a correct conclusion. The upper urinary passages are likely to be involved when the quantity of pus is very great—two or three ounces or more per day; when, with a large discharge of pus, the urine is only feebly ammoniacal; when the loins are painful on pressure; and the febrile movement and the decay of strength seem out of proportion to the vesical or urethral mischief; lastly, when the latter has been in existence for several years.

(*b*). *Pyelitis with tumour—pyonephrosis.*—Cases of this class do not usually present much diagnostic difficulty. There is an elastic fluctuating enlargement on one side of the abdomen, occupying the situation of a renal tumour, and a great discharge of pus with the urine. This discharge is apt to vary from time to time; and the dimensions of the tumour are observed to increase and decrease in inverse correspondence.

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* For the diagnostic signs of the sources of pus discharged with the urine see pp. 96, 97.

When the outlet from the sac is permanently sealed the nature of the lumbar tumour is much more obscure. It is liable to be mistaken for hydronephrosis, a hydatid cyst, a perinephritic abscess, or an abscess or cyst of the spleen,[*] or liver. The diagnosis, in such a case, turns first on the existence of a tumour presenting the physical signs of a renal tumour (see DIAGNOSIS OF CANCER OF THE KIDNEY); secondly, on the evidence of fluidity of its contents; and thirdly, on the signs that that fluid is purulent (recurrent rigors and hectic).

Prognosis.—The prospects of a patient suffering from pyelitis differ greatly according as one or both sides are affected, and according to the nature of the exciting cause.

Double pyelitis arrived at the purulent stage is a disorder of very grave consequence, whatever may have been its mode of origin, and usually proves fatal in the end. When the disease is confined to one side, the issue may be favourable, either with or without destruction of the kidney. Cases of this last class are not infrequent: numerous examples have been recorded, in which one kidney has been found, after death from some other cause, bearing the marks of previous sacculation and suppuration. Sometimes nothing is found in the situation of the kidney beyond the capsule of the gland tightly embracing a urinary calculus; in others, an empty cellular sac; in others, a sacculated pouch completely filled with concrete pus. An example is reported by Kussmaul[†] in which pyonephrosis was encountered (*post-mortem*) apparently in an early stage of obsolescence. The patient died of constitutional syphilis, with lardaceous liver and spleen, and Bright's degeneration of the left kidney. The right kidney was converted into a soft, thickwalled tumour, as large as a child's head, situated in the right hypochondrium. It was filled with thick, inodorous pus; the renal tissue had totally disappeared. The sac had contracted adhesions to all the surrounding parts. The ureter was adherent to the wall of the sac, so that the escape of the pus was prevented. A probe, however, could be passed along into the dilated pelvis. The disease was evidently of old date; no symptoms (beyond the physical signs of tumour) referrable to it were observed during life; and the cause of its production could not be clearly made out after death.

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[*] Caffe records a case of pyonephrosis in a Portuguese physician, residing in Paris, which was mistaken for a cyst of the spleen. No pus had ever appeared in the urine; indeed, there were no urinary symptoms at any time. Nelaton punctured the supposed cyst and withdrew 4½ litres of pus. The patient survived 55 days. (Gaz. des Hôp. 1855.)

[†] Würzb. Med. Zeitschr. 1863, p. 43.

The gravity of pyelitis has a close connection with the nature of its original cause. Cancerous and tuberculous pyelitis invariably prove fatal: the prognosis is almost equally hopeless when the disease is secondary to enlarged prostate, intractable disease of the bladder, or urethra. The prospect is more favourable, though still exceedingly grave, in cases which follow pregnancy, or depend upon renal gravel, calculus, or hydatids.

When pyelitis is secondary to some acute disease (zymotic fevers, etc.) it is of very slight consequence, and speedily passes away with the subsidence of the primary disorder.

Rupture of the sac into the thoracic or peritoneal cavities is speedily fatal. Rupture into the intestine generally, if not always, proves ultimately fatal; but the sac may open through the loin with a favourable issue; though this is exceptional.

Treatment.—The chief general indications in the management of cases of pyelitis are:—to remove the exciting cause, and, secondly, to arrest or control the purulent discharge.

When pyelitis is secondary to Bright's disease, diabetes, scurvy, purpura, diphtheria, typhus or other zymotic fever, the gravity of the primary disease so overshadows the secondary affection that the latter rarely demands separate attention. It is only in the rare hæmorrhagic examples when the loss of blood by the urine becomes threatening, that the internal administration of astringents and styptics becomes necessary.

The particular treatment applicable to the different species of pyelitis will be found described under the several headings of CONCRETIONS IN THE KIDNEY, PARASITES, TUBERCLE, CANCER, ETC.

The following observations will find their application in those cases, both acute and chronic, in which the inflammation of the pelvis and infundibula is a leading feature of the complaint and the source of the more important symptoms.

If the attack be acute, and accompanied with pain in the renal region, frequent and painful micturition, bloody urine, and fever, the loins should be cupped to eight or twelve ounces; the cupping should be followed up with warm baths and hot poultices to the loins. Warm diluents should be freely administered. Opium and other anodynes are sometimes demanded on account of the intensity of the suffering and evidence of spasm of the ureter.

In chronic cases, when the secretion of pus is profuse, the efforts of the practitioner must be directed to lessen the discharge, and to bring the renal tumour, if there be any, to a state of contraction or of obsolescence, and throughout to keep up the general health to the highest possible standard.

Among the remedies which are available to check the discharge of pus are, the mineral acids, alum, vegetable astringents, balsamic and terebinthine substances. These last are only applicable when the disease is thoroughly chronic, and a stimulant to the mucous membrane is required.

The metallic astringents have also been occasionally employed with success, when other means have failed. Mosler relates the following instance of the good effects of acetate of lead, in a case of uncomplicated pyelitis arising (presumably) from cold:—

David G., æt. 19, cutler, came under treatment in August, 1861. In the spring of the year he had been working in a very cold place, and his illness commenced with a smarting pain in passing water. This was followed by the appearance of pus in the urine. When the case came under observation, it was quite uncomplicated; the only complaint was smarting in making water, and a desire to void it about every hour. Compression of the urethra caused no pus to appear at the orifice; the pus was thoroughly mixed with the urine, giving the latter a turbid appearance. After standing, a layer of pus subsided to the bottom of the vessel, about half an inch thick. The microscope brought to view pus corpuscles and varied forms of epithelial cells, some of which were fatty. The albumen was no more than corresponded to the amount of pus. The reaction was acid, and continued so throughout. At first vegetable astringents in large doses (10 grains of tannic acid thrice daily) were employed ; then balsamic remedies in the form of Griffith's mixture, &c. ; then alkalies (sod. bicarb. ʒiii. daily). The quantity of pus remained stationary, in spite of all these remedies ; but the smarting in passing water had mostly ceased.

In the beginning of October, the patient complained for the first time of pain in the right lumbar region. At that time there were blood corpuscles in the urine, as well as pus and epithelium. The pus had increased. The alkalies were now combined with the use of warm baths ; the blood soon disappeared, and the pains ceased, but the pus continued undiminished.

On the 1st of January, 1862, the use of acetate of lead was commenced, in doses of three grains three times a day. At the end of eight days the dose was increased to four grains three times a day. The effect of the treatment on the amount of pus was marked ; on the tenth day the quantity was visibly diminished, and shortly afterwards it disappeared altogether. Some months later the patient presented himself again ; the urine was found quite free from pus, and the general health blooming. These large doses produced colicky symptoms toward the end of the second week ; and there was at the same time a decided, though not great, diminution in the daily quantity of urine.

The tincture of the sesquichloride of iron has sometimes proved of signal service, as in the following example :—

B. H., a woman æt. 51, was admitted under my care into the Royal Infirmary, in December, 1862, in a state of extreme weakness and emaciation. On examining the urine it was found acid, loaded with pus mixed with some blood. Micturition was frequent with smarting pain. Careful and repeated exploration of the bladder failed to detect a stone. The right kidney was painful on pressure, and the anamnesis disclosed obscure history of renal calculus. There was no fulness in the loin. The daily quantity of pus was estimated at three ounces. The deposit in the urine contained no cellular elements except pus and blood.

She was first put on a mild alkaline treatment, with generous diet, and six ounces of wine. No improvement followed ; she continued to lose ground, and was unable to leave her bed ; the tongue became dry at times, and symptoms of severe hectic showed themselves. The alkalies were then discontinued ; and 30 drops of tincture of steel in a wineglass of water, administered three times a day ; the wine was increased to 10 oz. This treatment was continued for many weeks, and gradual amendment set in. Blood disappeared wholly from the urine, and the discharge of pus was reduced to less than half an ounce. The general health improved proportionally ; and in March, 1863, the patient was able to leave the hospital in a fair way of recovery. She afterwards presented herself among my out-patients from time to time for some months, and steadily gained strength. At length she went to her work (weaving), and I heard nothing more of her until April, 1864. All her symptoms had returned in great severity some weeks before. She declined to comply with my recommendation to enter the Infirmary, and, four weeks after, I heard of her death.

Among the general means designed to keep up the vigour of the system, the most important are cod-liver oil, quinine, nourishing diet, and, above all, change of air. Sea-side localities are preferable, and even sea-bathing may be recommended, if the patient's strength permit.

When renal tumour exists, it is not, as a rule, advisable to take any steps with a view to procure evacuation of the sac through the integuments, unless there be decided indications of pointing. It must be remembered that there is always a chance (supposing the disease to be confined to one side) that, with rest and patience, the pus may become inspissated, and the abscess pass into a permanently obsolescent state; or that gradual emptying of the sac may take place with final atrophy of the renal tissue. The advantages of an expectant treatment are strikingly illustrated in the following case, recorded by Henninger:—

The patient received a blow on the left lumbo-renal region in 1848. Obscure, persistent, renal pains followed the accident. Three years after, the patient had nephritic colic on the left side, which recurred in periodical paroxysms, resembling ague. In 1852, the attacks recurred about every three days; they were followed by the discharge of a highly purulent urine. Mixed with the pus were found epithelial cells and crystalline deposits. A tense elastic tumour was discovered in the left hypochondrium, extending as far as the vertebral column. After a paroxysm, and discharge of pus with the urine, this tumour was only doubtfully perceptible, but in exploring along the course of the ureter, a body as large as a nutmeg was discovered in the iliac fossa, in the track of the ureter. The nature of the case was now clearly made out to be calculous pyelitis with tumour. The advice of M. Schutzenberger was to establish a fistulous opening in the renal region with a view to provide a safe outlet for the pus, and thus relieve the neuralgic paroxysms. On consulting with Prof. Sedillot it was agreed to wait the progress of events, in the hope that the renal tissue would be gradually absorbed, and the kidney reduced to a membranous pouch, which, on the cessation of the secretion of urine, might eventually contract. These hopes were realised. A merely palliative treatment was adopted; and six months afterwards the patient saw an end to his sufferings; he has continued since in uninterrupted health.*

There are cases, indeed, in which the distension of the sac becomes so great, that the peril of rupture into the peritoneum, exceeds the risk of making an opening through the integuments. In such a contingency, the same rules apply as for the puncture of serous cysts of the kidney (see HYDRONEPHROSIS—TREATMENT); the opening should be made in the lumbar region, where the sac bulges most, and where danger of wounding the peritoneum is least.

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* Henninger. *Thèse de Strasbourg*, 1862.

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CONCRETI

RAYER—l. c. T. iii., p. 10.
VIRCHOW—Gesammelte Abha
ROSENSTEIN—l. c., p. 417.
VOGEL—Krankh. d. Harnberei
LEROY D'ETIOLLES (fils)—Trait

CLOSE examination of s
the existence of numerous
from the papillæ toward t
due to the precipitation of
canals. This is generally
cooling of the body after
the urates, and causes the
tubes.

In infants dying within
striæ are almost invaria
been found in still-born
(Hoogeweg and Martin).

A similar precipitatio
and constitute the first li

quently, they are rolled down along the ducts by the stream of urine, and deposited in the infundibula and pelvis of the kidney; and even many thousands of minute calculi, formed in this manner, may be encountered after death, in these situations (see case of J. R. p. 334). Agglomerations of larger size may begin in the same way, or the precipitation may first occur in the infundibula and pelvis.

In number, size, and shape, renal concretions present the greatest diversities. A kidney may contain only one concretion, three or four, or several hundreds. In size they vary from a pin's head, or a hemp-seed, to a horse-bean; and if a concretion become permanently lodged in the pelvis or its appendages, it may go on increasing to a weight of several drachms or ounces. Such a calculus is usually moulded to the divisions of the pelvis, and assumes various grotesque, branched, or arboraceous, forms.

The *anatomical changes* produced by renal concretions, are, congestion of the kidneys, abscesses, pyelitis, pyonephrosis, and hydronephrosis. These are considered under their respective headings.

Symptoms.—The existence of concretions in the kidney is usually indicated by an aching pain in the loins, occasionally rising into violent paroxysms (nephritic colic). This pain is characterized by its tendency to shoot along the course of the ureters down to the testicles and the inside of the thigh; it is also commonly attended with a sense of faintness, nausea, or even vomiting. The urine, in these cases, is voided with undue frequency, often with pain at the end of the penis, and it is apt to contain blood, pus, and epithelium from the pelvis of the kidney.

The colicky paroxysms are determined by dislodgement of the concretion from one of the infundibula into the cavity of the pelvis, or from one part of the pelvis to another; but the most severe attacks are caused by the impaction of it in the ureter.

The descent of a calculus along the ureter into the bladder is productive of very distinctive symptoms. The patient is suddenly seized with intense pain in the region of the affected kidney, accompanied with a sense of deadly faintness, cramp and sickness. The pain radiates in various directions, but chiefly along the ureter to the bladder, scrotum, end of the penis and the inside of the thigh. The testicle is retracted; there is incessant desire to make water, but the flow of urine is either partially or wholly suppressed. In the former case, the urine is high coloured, often mixed with blood, and voided in drops with burning pain. Violent and frequent vomiting follows; the skin is covered with a cold sweat; there is constant restlessness; the patient tosses from side to side, and assumes in succession a score of different positions in the hope of relief. If the symptoms are not speedily relieved, a febrile movement is produced, which, sometimes, attains a high degree, with hot skin, quick pulse and incessant thirst.

After these symptoms have continued a certain time—it may be hours, it may be days—relief comes, often quite suddenly. The patient feels something drop into the bladder, and, all at once, his agony is past. Sometimes, however, the concretion fails to clear the ureter, and becomes impacted in some part of its course. In this case, the subsidence of the symptoms is more gradual, and less complete. In other, fortunately still rarer, instances the opposite ureter has already been rendered impervious by the impaction of a calculus on some previous occasion, and the blocking-up of the hitherto open channel is followed by total anuria, which leads to a rapidly fatal issue, with or without uræmic symptoms.

Renal calculi are sometimes wholly latent. They may even attain a large size, and destroy extensive portions of the gland, without betraying their presence by a single symptom. Or, again, renal symptoms may exist for a longer or shorter period and then wholly and finally cease. This latter event may occur under two circumstances: either the concretion completely occludes the ureter, and determines gradual atrophy of the kidney, or it becomes encysted in a lateral pouch or diverticulum, and ceases to impede the flow of urine and to irritate the mucous membrane.

The *diagnosis* of a calculus, or calculi, in the kidney or pelvis (except in latent cases) is not generally attended with much difficulty. The locality distribution and paroxysmal recurrence of the pains, with the pyelitic characters of the urine, are usually sufficient to indicate the cause of suffering. Neuralgia of the lower intercostal and abdominal nerves sometimes presents great severity, and a paroxysmal character. It is distinguished from renal colic by the absence of blood, pus, and transitional epithelium in the urine. More difficult to distinguish are those cases in which nephritic colic is produced by the impaction of blood-clots or hydatids in the ureter; indeed, absolute certainty cannot often be obtained in these cases until the appearance of gravel, hydatids or clots in the urine sets the question at rest. The antecedents of the patient sometimes throw an important light on the diagnosis, and a knowledge of the nature of a foregoing attack will furnish a key to an existing one.

In the absence of colicky paroxysms—where the symptoms consist only of obscure lumbar pains and slight disturbances of micturition, careful and repeated examination of the urinary deposit becomes the principal means of arriving at a precise diagnosis. If the symptoms be due to calculus, the deposit will, in all probability, contain scattered blood-disks and spindle-shaped, tailed and irregular epithelial cells from the upper urinary passages. These may be accompanied with pus corpuscles, and minute agglomerations of uric acid, dumb-bells of oxalate of lime, or some other form of calculous deposit. These unnatural conditions of the urine are intensified by violent exercise, and diminished or altogether suppressed when the patient maintains a state of rest.

The *treatment* of renal concretions must be modified according to the existing symptoms and the anatomical changes which may be inferred to have taken place in the kidneys.

During the paroxysms of renal colic, the remedies indicated are warm baths, emollient enemeta, cupping the loins, and, in highly sthenic cases, venesection. The dolorous spasm of the ureter must be combated by free administration of opium. This drug is freely tolerated in cases of this class, and full doses should be repeated until the system is plainly brought under its influence. When the irritability of the stomach is such as to prevent the absorption of the drug, it should be introduced per rectum or by subcutaneous injection. Belladonna may be substituted where opium disagrees. The secretion of the urine should be encouraged by warm demulcent drinks; hot poultices should be applied to the loins or abdomen, as the local symptoms indicate.

Change in the position of the patient sometimes suffices to dislodge a calculus which lies upon, but has not become fully engaged in, the orifice of the ureter. Manipulation of the abdomen in the course of the ureters may also facilitate the descent of the concretion. Dr. Simpson witnessed relief follow complete inversion of the body.*

In the intervals of the nephritic attacks, or when none exist, the treatment must be conducted either with a view to dissolve the concretion (see SOLVENT TREATMENT OF URINARY CALCULI) or according to the rules laid down for the management of chronic pyelitis. When abscesses form, or pyo- or hydronephrosis is established, the modes of treatment described under these headings must be followed out.

Incising the kidney through the loins, and extracting the offending calculi through the wound (nephrotomy), is a method of treatment as old as the time of Hippocrates. It is, however, not recommended by modern surgeons, except when suppuration has taken place, and the abscess is manifestly pointing in the loins. When such an abscess is opened, exploration should be made with a probe, and if concretions are detected thereby, cautious endeavours may be made to remove them by suitable instruments (Hevin and Velpeau—Oldfield, Thèse de Paris, 1863).

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* Edin. Med. Journ. 1858–9, p. 76.

CHAPTER VIII.

HYDRONEPHROSIS.

Glass—Phil. Trans. 1747.
Johnson—Monthly Med. Chir. Journ. 1816 (July).
König—Krankheiten der Nieren. Leipzig, 1826, p. 152.
Rayer—Maladies des Reins, tom. iii. p. 476.
Lee—Med. Chir. Trans. xix. 238.
Hare—Med. Times and Gaz. 1857, i., 29.
Kussmaul—Würzb. Med. Zeit. Bd. iv. Heft. I.
Stadfeldt—(Etiology of) Monatsschr. f. Geburtsk. 1862, p. 69.
Farre—Lancet, 1861, ii., 472.
Rosenstein—Nieren-Krankheiten, 350.
Dumreicher—Weiner Med. Halle, March 27, 1864.
Strange—Beale's Archives, vol. iii.
See also :—Path. Soc. Trans., vii. 262, 263, 265; ix. 318; xiii. 128, 137, 147, 151; xiv. 195.

When any impediment exists to the flow of urine from the kidneys the secretion accumulates behind the obstruction and distends the parts above. The first effects of the pressure of the accumulated urine are felt in the hither portions of the ureter and the pelvis of the kidney; these parts become dilated. Then the renal substance is compressed, and becomes partially or wholly atrophied and absorbed; so that the organ is at length hollowed out into a pouch or bag, consisting of the fibrous capsule of the kidney. When these changes are associated with suppuration of the lining membrane the condition termed pyonephrosis (already described) is produced. But in a considerable number of instances the obstruction is unaccompanied with purulent formation; the distension proceeds painlessly and gradually. This is the case when the impediment arises from some congenital malformation; also when it is incomplete, or is established by degrees. To this condition the names of "dropsy of the kidney" and "hydrorenal distension" have been applied; but both designations have given place to the term hydronephrosis, introduced by Rayer, and now generally adopted.

Morbid Anatomy.—Some years ago I exhibited to the Manchester Medical Society a typical example of what may be called a fully developed hydronephrosis. It consisted of a large membranous bag, 13 inches long by 8 inches broad. It represented the right kidney of a woman, who, during life, was supposed to be the subject of ovarian dropsy. She had been twice tapped under that impression, and died of peritonitis after the second operation. It proved, after death, to be the right kidney and pelvis monstrously dilated. When filled with fluid the cyst had a lobed or sacculated exterior, like an enormous colon. The ureter was incorporated with the posterior wall of the cyst; and opened obliquely into the dilated pelvis, with a valvular arrangement resembling that at the entrance of the ureter into the bladder. The channel was pervious to a probe; but the valve-like deformity of its orifice (evidently congenital) prevented the free escape of urine.

On cutting open the cavity a complete fibrous skeleton of the kidney was disclosed (see diagram; Fig. 47). The pelvis was dilated to the size of a large cocoa-nut, and formed a sort of *atrium*, in the interior of which seven smooth rounded openings were situated, large enough to admit the little finger. Each of these openings led into a rudely pyramidal chamber, the bulging base of which corresponded to one of the external lobulations. These chambers were separated from each other by strong membranous septa; but they communicated freely with each other through the openings into the enlarged pelvis. Not a particle of kidney substance existed in any part; but three flattened fibro-cartilaginous nodules were found imbedded in the outer wall of the sac. The fibrous membrane which composed the pouch and septa was exceedingly tough and strong, much resembling the dura mater. The outer membrane evidently consisted of the thickened and hypertrophied tunica propria, and was continuous with the fibrous structure of the dilated pelvis. The septa corresponded to some of the embryonal divisions of the kidney; and the circular openings represented the chief divisions of the pelvis.

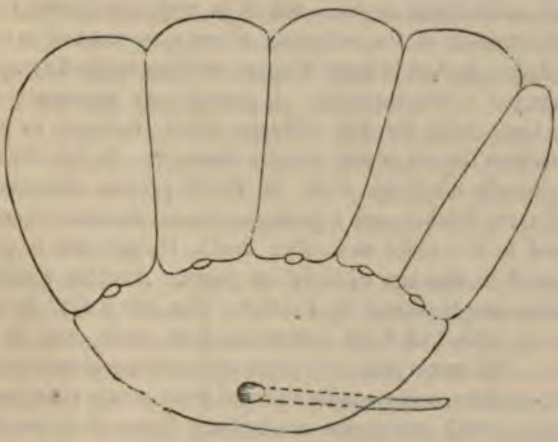

FIG. 47. Diagram of a fully-developed hydronephrosis.

From this type there are many variations. The sac may not be nearly so large: it may not exceed the dimensions of the healthy organ: it may be even smaller. The chambers vary much in depth, and in number; there may be only two or three; or the whole sac may consist of only a single cavity. The cyst may be composed in varying proportions of expanded pelvis and dilated kidney: sometimes the expansion is almost confined to the former, which is transformed into a globular swelling occupying the hilus of the kidney. The absorption of the secreting tissue is not usually complete. The stagnating urine exerts its pressure in the first instance upon the papillæ, which become flattened, and, as it were, effaced; then the bodies of the pyramids are compressed and gradually atrophied; lastly the cortex is encroached on, more and more, until it is reduced to mere islets of reddish tissue on the membranous parietes; and at length, if life be sufficiently prolonged, these disappear, and not a vestige of the glandular tissue remains.

When only one kidney is involved, a compensating hypertrophy of the opposite organ takes place, and the urinary function goes forward unhindered so long as the latter continues sound, and its channels of excretion free. There is nothing astonishing in this; but it is very unexpected to find that destruction of the secreting tissue may proceed to an extreme degree in *both* kidneys without evoking marked symptoms of deranged urine-secretion. A person may apparently exist for a time with the two kidneys wholly reduced to membranous sacs devoid of any tubular structure. In Dr. Strange's case, already cited (p. 153), in which profuse diuresis had existed from infancy, not a particle of renal substance could be detected in the renal sacs after death, though life had been protracted to the age of eighteen years. Another equally remarkable case is related by Faber.[*] The subject of it was a little boy, who had been ventricose from birth, and in weak health. The urine generally presented nothing abnormal; but on two or three occasions he suffered from severe paroxysms of strangury, with symptoms resembling those of stone in the bladder. Notwithstanding these drawbacks the boy was in better health in the last year of his life than he had been for the previous four years, and was able to go about. When he had reached the age of 5½ years he fell from a chair and died suddenly in consequence. The autopsy revealed the following state of the urinary organs. Both kidneys were converted into large pouches or sacs, containing no trace of kidney substance. The renal pelves were likewise greatly distended, and the ureters so completely resembled the small intestine that the dissector held them several times in his hand in the belief that they were a coil of intestine. The bladder contained a little turbid urine; its walls were greatly thickened. There was no disease of the prostate, neck of bladder, nor urethra. The entrance from the bladder into the dilated ureters was sufficiently open.

In these and similar cases the atrophy of the secreting tissue had doubtless been going on slowly, and progressively, from the time of birth. It cannot be assumed that complete sacculation of the kidneys and total absence of renal tissue

[*] Würt. Correspondenz-Blatt., Bd. xii, 266.

existed from birth; for, as was pointed out by Rayer, infants with congenital double hydronephrosis are not viable. Life is probably eked out in such cases by the vicarious activity of the skin and bowels, which undertake some portion of the depurative functions properly belonging to the kidneys. Death commonly takes place, in cases of this class, quite suddenly—sometimes with violent uræmic phenomena.

Of 37 cases collected by me, the hydronephrosis was confined to one kidney in 26 instances, and affected both (double hydronephrosis) in 11 cases. When the hydronephrosis was single the right side was more frequently affected than the left (16 right, and 10 left).

Hydronephrosis sometimes attains enormous dimensions; and fills the abdomen with a soft fluctuating intumescence, reaching from the borders of the ribs to the pubes. Rayer cites an instance in which sixty pounds of fluid were withdrawn from the sac. But the most extraordinary example which I have discovered is the following, related by Mr. Samuel Glass in the Philosophical Transactions for 1747:—

Mary Nix had been remarkable all her life for the preternatural size of her belly. Her mother stated that her daughter was born dropsical; but otherwise she proved healthy; and, notwithstanding the steady increase in the size of the abdomen, she lived to be near 23 years of age.

She is described as a tall and well-proportioned woman, except for the enormous size of her belly; and, for one of so unwieldy a bulk, to have been brisk and active. The menses, which appeared at the usual time of life, continued regular until within eight months of her death. The only complaint was of a pain occasionally felt in making water.

On the suppression of the catamenia, there succeeded a certain amount of dyspnœa, loss of appetite, and emaciation, with swelling of one of the legs, and ulcerations. These symptoms gradually increased until her death.

On taking the dimensions of her body before dissection, the circumference of the abdomen was found to be just six feet four inches, and from the xiphoid cartilage to the os pubis it measured four feet and half an inch! The cutaneous vessels distributed on the abdomen were remarkably large.

The thorax being laid open, the diaphragm was observed to be forcibly protruded into that cavity. The base of the heart lay under the right clavicle, and its apex on the most convex part of the diaphragm; which convexity advanced as high as the third rib. The lungs were surprisingly small, scarcely exceeding in magnitude those of a new-born child. When the abdomen was opened a vast cyst was displayed, from which 30 gallons of a light, coffee-coloured, limpid, fluid was withdrawn. The fluid was not in the least fetid. In figure, colour, thickness, and magnitude, this enormous bag very much resembled the uterus of a cow at the end of gestation. The whole inside was scabrous, and looked as if parboiled, and here and there was observed a small quantity of a coffee-coloured sediment. On the left interior part was discovered the orifice of a duct (ureter) which opened obliquely into the cavity of the sac, and would easily admit a large goose-quill. From this opening the tube advanced about twelve inches between the membranes of the bag obliquely upwards, and towards the right, from whence it was deflected downwards, and passed between the fold of the broad ligament into the bladder. The abdominal viscera were thrust aside in various directions. The left kidney and ureter were healthy.

The fluid contents of hydronephrotic cysts are generally altered urine. Urea, uric acid, as well as the alkaline and earthy urinary salts, have been found therein. Prout detected urea and uric acid in the contents of a double hydronephrosis from a still-born infant. Generally speaking, the fluid is much more watery than ordinary urine; and sometimes the organic urinous matters only exist in traces. The fluid may be variously coloured; it may contain a little blood, pus, and epithelium, and it is nearly always more or less albuminous.

In two cases the contents of the cyst consisted of a substance resembling colloid. The first of these is described by Dickinson:—

The patient was an old woman of seventy. For twelve years she had perceived a tumour in the left hypochondrium, which at length filled the belly. Constipation alternated with diarrhœa. The patient stated that she occasionally passed "nasty stuff" by the urethra, and that the tumour diminished in size for a time after that occurrence. She at length died of pneumonia. The left kidney was found converted into a large sac about a foot long, divided by septa into compartments. These compartments were filled with a gelatinous substance, which, under the microscope, presented the usual appearances of colloid matter. It lay, however, quite loose in the cyst, altogether unattached to the parietes. There was no obstruction whatever found in the ureter, nor in any part of the urinary channels. Dickinson supposed that an obstruction—probably from a calculus—existed at some previous period, which led to sacculation of the kidney; and that the colloid matter was deposited subsequently. (*Path. Soc. Trans. Vol.* xiii., *p.* 137.)

The second case is reported by Prof. Dumreicher of Vienna, and is remarkable in many ways:—

A girl of 13 had observed a swelling in the abdomen from her tenth year. This grew to an enormous size; the circumference of the abdomen, which was uniformly distended, measured 44 inches. The percussion sound was dull, except over a space of four square inches on the left side below the navel. Fluctuation was perceived over the swelling. Prof. Skoda, under whose care the girl first came, diagnosticated an ovarian cyst; but he pointed out the possibility of a hydronephrosis. The case then passed to the care of Prof. Dumreicher, who, on account of the dyspnœal distress, punctured through the abdominal wall, and withdrew 18 quarts of a colloidal brown-coloured fluid. The circumference of the belly now fell to 30 inches, and the relief to the patient was great. Six weeks later, 16 quarts more were withdrawn; and an injection composed of one ounce of Tincture of Iodine, in 4oz. of water, with a drachm of Iodide of Potassium, was introduced into the cyst. This proceeding proved of no effect. In about a month 14 quarts more were evacuated, and the injection repeated. Severe iodism followed, and continued for a couple of days. The patient then rapidly improved, and left the hospital. In about three months she returned, larger than ever. The belly now measured 46½ inches, and the breathing was much embarrassed; the heart's apex beat in the third interspace. In the course of the succeeding five months the patient was tapped four times, and an aggregate quantity of 37 quarts of fluid were withdrawn—making a total, from the beginning, of 85 quarts! The fluid changed character as the tappings were repeated: it became more and more mixed with blood, and at length with pus. On one occasion a drachm of the crystallized sesquichloride of iron, dissolved in six ounces of water, was injected. This was followed by severe symptoms. At the last, a fistulous passage into the cyst was kept open by an elastic catheter, through which the cyst was evacuated twice daily, and washed out with warm water. Notwithstanding these precautions, the contents of the cyst grew daily more foul, and the patient's strength steadily diminished. She died after having been under observation about a year. On opening the belly the cyst was found to be the right kidney enormously dilated. The sac was intimately adherent to the liver; and the right lobe of the latter was so compressed that it was reduced to half the size of the left lobe. The cæcum and the end of the ileum were fixed by adhesions to the front of the cyst; the rest of the bowels were thrust into the left hypochondrium. When opened, the sac was found in some places thin, in others several lines thick: they were divided into compartments, of which the parietes were traversed by broad membranous bands in various directions, which divided the cavities into a number of small loculi. In these latter a number of cysts with yellowish contents were situated. The anatomical cause of the distension was not very clearly made out; but it appeared to consist in a congenital obliquity of the origin of the ureter, whereby a valvular condition was induced, which impeded the flow of urine. The ureter, after its origin in the cyst, ran in a half circle, downwards and backwards, intimately adherent to the cyst walls, and compressed by them. A small supernumerary renal artery arose from the aorta a few lines below the principal branch. The left kidney was enlarged, but healthy. (*Wiener Med. Halle*, 1864, *p.* 139.)

Etiology.—The anatomical conditions which lay the foundations of hydronephrotic distension of the kidney are exceedingly varied. Out of 37 cases which were collated for the purpose of the present article, there existed congenital malformation in 14 cases—affecting the kidneys, the ureter, or the renal artery. In two of these, a supernumerary renal artery crossed and compressed the ureter near its origin; in four, the ureter was congenitally imperforate; in three, the ureter entered obliquely into the pelvis of the kidney, creating a valve-like impediment, which necessarily increased as the pelvis expanded. In a case recorded by Dr. Hare a very curious deformity was found in both ureters, which he thus describes:—"On taking the mass (the dilated kidney) in the hands, and pressing very firmly, no fluid escaped by the ureter; examining into the cause of this, it was found that the ureter, at a little distance from its origin, was coiled on itself—like a turn and a half of a corkscrew brought closely together, and that this coil was adherent to the lower part of the dilated pelvis; above this part, the ureter was slightly dilated; below it, not at all. The coils just mentioned acted as a valve-like obstruction to the course of the urine, for on gently dissecting away, with the point of a scalpel, the tissue which held the coils together and united them to the tumour, the retained fluid rushed readily out by the end of the ureter in a full stream."*

In 8 out of the 14 congenital cases, the hydronephrosis was double—that is, it affected both kidneys. Two of these perished still-born, and a third died in thirty hours after birth: but Dr. Hare's patient (just mentioned) survived to the age of thirty-eight years; and the remaining four lived for periods varying from five and a half to eighteen years. We must assume, in these latter cases, that the impediment to the urinary flow was at first incomplete (though the malformation was congenital), and that its effects were not fully developed until a subsequent period, and then probably with extreme slowness.

In an instance cited by Rayer, the obstruction (congenital) was constituted by an imperforate urethra: the bladder, ureters, and kidneys were distended into capacious sacs (l. c. iii. 504).

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* Med. Times and Gaz. 1858, I. 234.

Congenital hydronephrosis is often associated with malformations of other organs—imperforate anus, hare-lip, club-foot, &c.

Of the 23 cases in which the obstruction arose later in life, it was due, in eight instances, to the impaction of a calculus in the ureter; and a similar impediment, although not actually found, was inferred to have existed at some previous period in two others. In four cases, a narrowing or obliteration of the ureter existed near its origin or its termination, produced presumably by some past inflammatory or ulcerative process, followed by subsequent constriction. In six instances, the ureters were compressed near their entrance into the bladder by a pelvic tumour—gravid uterus, ovarian cyst, or a cancerous growth: cases of this class are no doubt much more frequent than these numbers indicate; but they are generally slight in degree, and seldom go on to the production of a palpable tumour in the flank.*

In a number of the cases collated, a mechanical cause for the distension could not be assigned, or, such a cause was only obscurely indicated. In some of these, no doubt, a more careful inquiry would have solved the difficulty; but still there are cases which must at present be regarded as mechanically inexplicable.

The following case by Boogaard illustrates in a striking manner how a congenital malformation, which, at first, scarcely offered any obstruction to the course of the urine, comes, step by step, to constitute a greater obstruction, and at length produces fatal results:—

A young man of twenty, otherwise in good health, had suffered, from time to time, from paroxysms of pain, followed by nausea and vomiting. On the 3rd of February 1857, he was seized with one of these paroxysms, accompanied with obstinate constipation. The vomiting became intractable; the vomited matters contained blood and sarcinæ; and no passage could be obtained by the bowels. On examining the abdomen, a doubtfully fluctuating swelling was detected in the right flank. The symptoms were attributed to an organic affection of the liver. Under a continuance of these symptoms death took place in five days.

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* Stadfeldt found dilatation of the ureter common in women dying in childbirth, even when there was no lateral displacement of the womb. In sixteen *post-mortem* examinations he found such a dilatation nine times; it almost always begins where the ureter crosses the common iliac. Hydronephrosis from this cause (puerperal) is much more frequent on the right than the left side. Out of twelve cases, Stadfeldt found it only once on the left. (Monatsschr. f. Geburtsk. 1862, p. 71.)

At the autopsy, a bladder-like tumour as large as the fist was found in the right hypochondrium, situated between the liver, the colon, and the duodenum: it was united by adhesions to the two latter. The colon was not constricted at the adherent spot; but the duodenum was so tightly stretched over the tumour that its calibre was almost effaced. The stomach was greatly distended, and filled with a dark-coloured fluid.

A closer examination of the tumour revealed the following:—It consisted of the pelvis of the right kidney, greatly distended. The right renal artery was abnormally distributed; it divided close to its origin into two branches, one of which ran to the upper, and the other to the lower part of the hilus. The lower branch crossed the ureter near its origin, and exercised a certain compression upon it. The enlarged pelvis pressed forward between the two branches of the renal artery, in such a manner that the origin of the ureter was drawn beyond the level of the lower renal artery, compelling the ureter to loop itself round this branch in order to reach the bladder. Thereto was added a third mechanical obstacle, namely, the adhesion of the ureter in the first part of its course to the outer surface of the distended pelvis, for the space of three quarters of an inch.

The enlarged pelvis contained ammoniacal urine, mixed with blood and mucus. The corresponding kidney was long and narrow, but otherwise healthy, and scarcely atrophic. The left kidney was natural. (*Arch. f. d. Holländische Beitr. z. Natur-und Heilk. Bd.* I., *p.* 196).

The explanation of these appearances seemed to be this:—First, the lower renal artery compressed the ureter, and prevented the pelvis of the kidney from properly emptying itself until a certain pressure was exerted on its walls by the accumulated urine. This impediment was intensified by the curving of the ureter round the lower renal artery. The pressure so exercised probably excited inflammation and adhesion of the ureter to the outside of the expanded pelvis, and again of the latter to the colon and duodenum. The symptoms during life were thus explained. The periodical attacks of nausea and vomiting depended on the periodical dilatation of the sac and the pressure of it on the duodenum. Evacuation of the sac, when the pressure of the accumulated urine reached a sufficient height to overcome the obstructions, caused the paroxysms to subside. In the last paroxysm the resistance proved more obstinate; the duodenum became altogether occluded—hence the constipation; and the portal vessels became probably implicated—determining effusion of blood into the stomach, and hæmatemesis.

Hydronephrosis arises under such a variety of anatomical conditions, that its general etiological relations offer, as might have been expected, little that is characteristic. No age is exempt—not even fœtal life; nor is any especially liable: the two sexes, in the cases collated by me, were found nearly equally represented—19 were males and 16 females; in two infants the sex is not mentioned.

The *symptoms* of hydronephrosis depend mainly on the nature of its anatomical cause and on the size of the sac. If the sac be small and the opposite kidney sound, symptoms may be altogether wanting; old age may be reached without suspicion that one of the kidneys has been changed into a membranous sac, and the anomaly may be first discovered at the autopsy.

Generally, however, the distension goes on to the formation of a palpable tumour in the abdomen; and sometimes, as we have seen, this tumour attains an enormous size. Setting aside the cases which perished still-born, or within a few weeks of birth, there existed among the 34 remaining instances 17 in which abdominal intumescence was detected during life; in 13 of these the tumour was confined to one side, in four a double tumour existed.

In its topographical characters a hydronephrotic tumour presents the general physical signs of renal tumour. The swelling is situated in the flank; it reaches backwards in the lumbar region to the spine, upwards into the hypochondrium, downwards into the iliac region, and forwards to the umbilicus—encroaching on these regions variously according to its magnitude. The colon is usually in front of it; and the small intestines are thrust into the opposite side of the abdomen. Of the several displacements of the organs on either side I need not add anything to what is detailed in the chapter on cancer of the kidney, where the general characters of renal tumour are fully described. The special characteristics of hydronephrosis are its soft undulating feel; an outline, which is sometimes distinctly lobulated; and the evidence of fluctuation. There is one peculiarity which is pathognomonic when present, namely, the sudden diminution or disappearance of the swelling coincidently with the sudden discharge of a large quantity of urine. This sign is not always available; but it is sufficiently frequently met with to give it an important diagnostic value. It occurred in 7 out of the 17 cases in which the existence of a tumour was clinically ascertained.

In Dr. Hare's case of double hydronephrosis, already alluded to, subsidence of the tumour from this cause took place on one occasion on the right side, on another on the left side; presenting a succession of events sufficiently puzzling even to an acute observer.

The tumour is usually quite painless, and unaccompanied by any inconvenience except from its bulk. Occasionally, however, tenderness exists over it; and the action of the bowels is irregular. When the dilatation arises from the impaction of a calculus, symptoms of nephritic colic occur at the time when the impaction takes place; or from time to time thereafter, if, as is most usual, some quantity of urine still continues to trickle past the calculus. Similar paroxysms are recorded in two instances where no calculus existed.

The state of the urine usually furnishes no information: in the great majority of cases it is natural; sometimes, however, it contains a little pus; but never in quantity. During the attacks of nephritic colic it may contain blood; and be discharged with great pain, retraction of the testicle, vomiting, etc. The history of these attacks sometimes yields an important clue to the nature of the case. When both kidneys are affected, symptoms indicating defective elimination of urine (uræmia) necessarily show themselves at length. A hydronephrosis implicating one kidney only may, as we have seen, cause little or no inconvenience for many years, even though its bulk be considerable. The opposite kidney performs a double duty and becomes correspondingly enlarged. An individual in this condition, however, leads an existence of considerable peril: for if anything happen to impede the function of the single kidney on which life depends, dangerous symptoms necessarily arise. Rayer supplies the following instructive example:—

M. V., æt. 64, had experienced, at the age of 22, pain in the right renal region, shooting obliquely toward the bladder in the direction of the ureter. This pain proved obstinate, and increased more and more; the urine was occasionally bloody, and sometimes of a dark colour; the patient became pale and thin. Little by little the urine ceased to contain blood, and re-assumed its normal characters; the general condition was perfectly restored; and for a long series of years M. V. enjoyed blooming health.

About the year 1820, M. V. began to grow stout; the belly became remarkably large; and latterly his great size considerably impeded progression.

On the 18th of September, 1834, M. V. experienced an uneasiness in the abdomen which constrained him to keep his bed; pains were felt all over the abdomen, but especially toward the region of the left kidney. This region was tender on pressure; the patient passed no urine; and the bladder was not distended. During ten days M. V. had no desire to void urine, and at the end of this period he only passed two glasses of a citrine colour. On examining the abdomen a voluminous tumour was detected, extending obliquely from the right hypochondrium to the left iliac fossa. Obscure fluctuation was felt in the tumour, which was considered to be formed by the distended right kidney (this was confirmed at the autopsy). The condition grew more and more serious as the suppression of urine continued—the tongue became covered with a slimy coating; the features altered; the nights were sleepless; the pulse failed; hiccup supervened, and the patient expired on the 13th of October, 1834.

On opening the body the right kidney was found prodigiously distended, and converted into a pouch filled with 7lb. 11 oz. of a viscid fluid; the tumour was 16 inches long from above downwards, and 7½ inches broad. The ureter was dilated at its origin, but soon underwent a sudden constriction; in this strangulated part a little calculus could be felt which had completely obstructed the duct. Below this obstacle, the ureter resumed its ordinary dimensions. The left kidney was considerably tumefied and reddened. The pelvis was notably dilated and covered with vascular ramifications; the left ureter, like the right, contained a small calculus lodged five inches below the pelvis. The bladder and other abdominal organs were healthy. The state of the right kidney explained perfectly the former ailments of M. V., and death was the consequence of the disabling of the solitary kidney on which his life had so long depended. (Mal. des Reins, t. iii., p. 490.)

Terminations.—Hydronephrosis may terminate in various ways. The obstacle may be dislodged, and the contents of the sac discharged, without subsequent re-accumulation. If, in such a case, a portion of the renal tissue be preserved, the organ will be enabled, in part, to resume its function. If the distension have been long established, and the secreting tissue extensively or totally absorbed, the organ after evacuation of the fluid shrivels up into an empty sac. These may be regarded as the most favourable modes of termination.

I find that out of 29 fatal cases, which supply information as to the cause of death, 12 perished from some other disease. Nearly all of these were slight, unilateral cases, which were latent during life. Three cases of double hydronephrosis perished still-born or soon after birth from abeyance of the urinary function. In 14 cases death took place at a later age as a direct or indirect consequence of the renal distension. Of these 14, one died wearied out with the bulk of the tumour and dysenteric diarrhœa caused thereby. Mr. Glass's patient died from pressure of the vast sac on the respiratory organs. Four cases, in which double hydronephrosis was established gradually, died from progressive abolition of the renal function —three of them with distinct uræmic symptoms. Two more, with single hydronephrosis, died from suppression of urine through impaction of a calculus in the opposite ureter. In three cases, repeated tapping was followed by suppuration of the sac and exhausting hectic. In another case, the second tapping was succeeded by fatal peritonitis. Pressure of the tumour on the adherent duodenum, and consequent intestinal obstruction, caused death in one instance (p. 415). It is remarkable that only in one solitary instance (to be presently cited) was death caused by spontaneous rupture of the sac.

Diagnosis.—The diagnosis of hydronephrosis is certain and easy only when subsidence of the tumour occurs simultaneously with a sudden excessive discharge of urine, or when trustworthy history of such an occurrence can be obtained. When this symptom is absent the recognition of the disease depends on the ascertainment of the existence of a fluctuating renal tumour, and the absence of the signs of suppuration.

Hydronephrotic tumours have most frequently been confounded with ovarian cysts, ascites, and hydatid cysts. From an ovarian cyst, hydronephrosis is distinguished by the presence of the colon in front of the swelling, and by the absence of a bowel sound on percussion in the corresponding lumbar region. Ascites is distinguished, when the hydronephrosis is single, by the existence of dulness in *both* flanks: but when the renal tumour is double, and both flanks are consequently dull as in ascites, the latter condition is recognised by the change of level assumed by the fluid when the posture of the patient is altered—dulness from dilated kidneys being fixed in its limits, however the position of the patient may be changed. A hydatid cyst is generally identified by the escape of hydatid vesicles with the urine, and sometimes by the presence of a hydatid fremitus. In the absence of these symptoms it may be quite impossible to establish the differential diagnosis of these two conditions by physical signs, and inferences must be drawn from the commemorative symptoms. It may be of use to remember, that while hydronephrosis is not unfrequently double, a hydatid cyst is scarcely ever so.

Pyonephrosis is distinguished by the purulent character of the urine—actual or historical—also by the existence of more severe constitutional symptoms, and especially of recurrent rigors. Circumscribed abscess of the kidney, and perinephritic abscess, are distinguished by their more acute course, the presence of pain and the signs of suppuration.

The *prognosis*, although necessarily grave, is less serious than in other kinds of renal tumour. When the affection is unilateral, not only may life be indefinitely prolonged, but there is always a chance that spontaneous evacuation of the sac may take place, or that the cyst may be punctured with success. If the opposite (hitherto sound) kidney show symptoms of deranged function, the gravity of the prognosis is immensely increased. When both kidneys are affected the issue is unavoidably fatal at length; but many years may elapse before the atrophy of the secreting tissue, or the completeness of the obstruction, reaches a degree incompatible with life.

Treatment.—If the disease be unilateral, and inferred to depend on the impaction of a calculus in the ureter, precautions should be taken against a similar occurrence taking place on the opposite side. The patient should be directed to keep the urine adequately diluted by systematic potation, especially on going to bed, and to avoid a too highly animalized diet.

In the absence of this indication an attempt may be made to overcome the obstacle, or, if that be impossible, to facilitate the passage of the urine past it. To this end, the tumour should be carefully manipulated or champooed, from time to time. As the swelling is usually painless, this can be accomplished without difficulty. In a little girl of eight, who came under my care in the Manchester Infirmary, this treatment seemed to be followed by success. She had a soft, obscurely fluctuating tumour on the left side of the abdomen, about the size of a child's head, which was considered to be hydronephrosis. This was diligently manipulated in every direction, with the aid of a lubricating ointment, on alternate mornings. After the third manipulation, she suddenly passed a large quantity of urine, and the tumour forthwith subsided, and did not again return so long as the patient continued under observation.

If evacuation cannot be obtained in this manner, further interference is not justified unless the expansion of the sac be such that its pressure threatens serious mischief. Under these circumstances tapping may be resorted to. The following case, related by Mr. Thompson of Nottingham, furnishes an example of the successful adoption of this plan; and the reasons set forth by the writer for the selection of the spot chosen for puncture seem to deserve attention. This case is likewise the solitary instance I have discovered, in which death was caused by bursting of the sac into the peritoneum:—

The patient came under Mr. Thompson's observation in May, 1851. He was at that time suffering from great pain in the region of the left kidney. There were considerable enlargement and tenderness on pressure extending over the left hypochondriac, lumbar, and iliac regions. Dulness on percussion also existed in these regions. Symptoms of nephritic colic had existed for a considerable period. Similar symptoms had been observed on a previous occasion, which were suddenly relieved after passing, all at one time, more than a chamber-pot full of water, of the colour of port wine. On the present occasion, a similar event took place; in about a week the sac had entirely emptied itself through the ureter and bladder. The symptoms disappeared, and the patient apparently soon recovered. In November he began again to suffer from the same symptoms, which increased up to Jan. 27th, 1852. At this time the side was greatly enlarged and tender. There was an obscure sense of fluctuation, and the dulness extended to the right as far as the linea alba; backwards to the spine; downwards to the lowest part of the iliac fossa. The organs in the chest were displaced upwards. The patient's sufferings were now so great that it was determined to draw off the fluid with the trocar. There was no doubt that the sac containing the fluid was a dilated kidney, or a cyst connected with the pelvis of that organ; and in either case Mr. Thompson was disposed to select the interval between the two last (floating) ribs near their anterior extremities at which to introduce the trocar. Mr. T. fixed on this spot for the following reasons:—

1. Supposing the fluid to be contained in a sac having communication with the pelvis of the kidney, the kidney would lie behind the sac, partly upon the last two ribs, and partly upon the quadratus lumborum muscle, its normal situation upon this side; and if the instrument were introduced at the place indicated, and its point directed a little forward, it would penetrate the sac without any risk of wounding the kidney.

2. If the sac consisted of a dilated kidney, the point selected would still be the best, as it would be near the part at which the organ began to dilate.

3. It would be behind the peritoneum, and therefore there would be less risk of wounding that membrane.

4. If the patient had been tapped in front, the trocar must have passed through the peritoneum twice: first, that portion lining the abdominal muscles, and second, that in front of the sac; and, supposing no adhesion to have taken place between these two parts, when the instrument was withdrawn, some of the contents of the sac might have escaped into the cavity of the peritoneum and given rise to inflammation. Besides, there would have been more danger of wounding some of the bowels, should any portion have become adherent by inflammation between the walls of the abdomen and the sac.

The operation was therefore performed between the two last ribs near their extremities. An incision was made through the integuments and muscles; a small exploring trocar was then introduced; and as there was evidence of the existence of fluid, a larger instrument was inserted, with its point directed slightly forward, and eight quarts of dark-coloured fluid were drawn off. It was a singular fact (which was explained on examination of the specimen after death), that soon after this fluid was removed, the further contents of the sac flowed in the natural direction along the ureter.

The patient soon recovered; but it was necessary to repeat the operation in December, 1852, when 3½ quarts of fluid were extracted; soon after which, as before, the sac emptied itself through the natural passages.

The patient soon got well, and did not require the operation again until near eight years afterwards (March, 1860). At this time seven quarts were taken away; not long after which the fluid again found its way along the natural passages, and the patient again made a quick recovery, and remained well until Sept., 1861. When then seen by Mr. T., he was suffering from his old symptoms. On the 5th of October he was suddenly seized with pain in the abdomen, with difficulty of micturition, cold sweats, rapid pulse, and an anxious countenance. He went on pretty well until the 10th of October, when he suddenly became worse and died.

The *post-mortem* examination revealed intense peritonitis. Three pints of dark-coloured water (resembling that found in the sac) were removed from the right hypochondriac and epigastric regions. The sac proved to be the distended left kidney—it contained four pints of fluid; a hole was discovered toward the left side anteriorly, where the rupture had taken place. The descending colon lay before and toward the left side of the sac; the ureter entered the cavity of the sac obliquely through the wall of the cyst. This obliquity of the entrance of the ureter offered a probable explanation of the closure of that tube when the sac was full, and the open state of it when the sac was empty. The rupture doubtless took place on the 5th of October, when the peritonitis began; and in all probability there was some escape of fluid, but not much, at that time; a further and larger escape took place on the 10th, after which the patient rapidly sank. There was no stone found in the bladder, nor any obstruction in the lower course of the ureter. (*J. Thompson, Path. Soc. Trans.*, vol. xiii.)

The early history of the case caused Mr. Thompson to surmise that the patient had formerly voided urinary calculi; but none were ever found. It is quite as probable that the obliquity of the entrance of the ureter into the pelvis of the kidney was a congenital malformation, and that this constituted the real cause of the hydronephrosis.

Dr. Hillier relates another case of congenital hydronephrosis repeatedly tapped, in front, with success:—

The patient was born with great enlargement of the abdomen, simulating ascites, for which it was mistaken till he was nearly four years old. It was then ascertained to be an enormous cyst springing from the right lumbar region. From its great size it caused difficulty of breathing and prevented his walking. The cyst was tapped in front, and 102 fluid ounces of clear non-albuminous fluid were drawn off, having all the characters of dilute urine. The fluid rapidly re-collected, and on a second tapping was found to be albuminous and purulent, but still to contain a considerable quantity of urea. Attempts were made to establish a permanent fistula anteriorly, and then posteriorly; but on each occasion the fluid after a time ceased to flow. Much irritation and depression followed the several tappings, so that the patient's life seemed to be endangered. After one of the operations a quantity of fluid was passed from the bladder exactly similar to that from the cyst, and quite unlike what was usually passed from the urethra; a temporary communication thus obviously being established between the cyst and the bladder. When the case was reported, the patient had been left without operation for some months, and had regained his strength; but the cyst remained, varying from time to time in size, and his urine was often purulent and fetid. It was presumed that there was some congenital malformation of the right ureter which rendered it liable to occlusion, but admitted, under some circumstances, of the passage of fluid. (*Brit. Med. Journ., April 8th*, 1865.)

CHAPTER IX.

CYSTS AND CYSTIC DEGENERATION OF THE KIDNEYS.

Rayer—Mal. des Reins, t. iii., p. 507.
Bright—Memoirs on Abdominal Tumours (New Syd. Soc.), p. 208.
Hawkins—Med. Chir. Trans., vol. xviii., p. 175.
Coote—Med Times, 1851, ii., p. 197.
Virchow—Gesammelte Abhandlungen, pp. 837, 864.
Siebold—Monatssch. f. Geburtskunde, 1854.
Beckmann—Archiv. f. Path. Anat., ix., p. 221.
Förster—Pathol. Anat., p. 357.
Path. Soc. Trans.—1848—9, p. 74; 1850—1, p. 131; 1851—2, pp. 377, 379, 384; iv., pp. 193, 199; v., p. 183; vi., p. 267; ix., pp. 309, 334.

Cysts are found in the kidneys under four practically different circumstances, namely:—1. Scattered cysts in kidneys otherwise healthy. 2. Disseminated cysts in the atrophic form of Bright's disease. 3. Congenital cystic degeneration. 4. General cystic degeneration in adults.

1. *Scattered cysts in kidneys otherwise healthy.*—It is not uncommon to find on the surface of healthy kidneys one or more cysts, with delicate walls, varying in size from a pea to a marble or a walnut. One or more of similar appearances may also be found in the interior of the gland, chiefly in the cortical substance. Such cysts are filled with a yellowish albuminous fluid—usually diffluent, sometimes gelatinous,—containing phosphates and carbonates, sometimes a large quantity of cholesterine, and very rarely urea and uric acid.

Cysts of these dimensions do not produce any symptoms during life; and their effects on the function of the gland is insignificant. Sometimes, however, cysts of this class attain a monstrous size, and form a tumour recognisable during life. Mr. Cæsar Hawkins gives an account of a remarkable case in which the right kidney of a boy, six years of age, had an enormous cyst attached to it. The cyst filled the entire right side of the abdomen from the false ribs to Poupart's ligament. The attached kidney was healthy in its structure, and the ureter free. In the wall of the cyst, separated by a distance of five inches from the kidney, there was inserted a small mass about the size of a walnut, which projected into the cavity of the cyst. This body proved to be a third kidney, consisting of a single lobule, with the cortical and tubular part perfect; and having a single mammillary process and calyx; but no excretory duct could be traced. The urine had been natural. The cyst was punctured during life: and about five pints of fluid were found in it after death. The fluid contained neither albumen nor any of the special urinary ingredients.

Dr. Hare (Path. Soc. Trans., vol. iv., p. 199) describes a very similar cyst taken from a man aged sixty-two. A tumour was detected during life on the right side, stretching from the ribs to the pubes. After death, a large cyst was found connected with the right kidney. The lower half of the gland was partly spread out over a portion of the tumour, and partly absorbed. The upper half was healthy; nor did it (nor the opposite kidney) contain any other cyst, with the exception of one, about as large as a hempseed. The large cyst contained an almost transparent pale yellowish-green fluid, quite limpid and diffluent when the cyst was first opened: but after the fluid had been exposed a few minutes to the air it set into a tremulous jelly.

In neither of these two cases was the pelvis of the kidney or ureter dilated. In both cases the disease was doubtfully traced to external violence.

2. *Disseminated cysts in the atrophic form of Bright's kidney.*—These have already been noticed in connection with Bright's disease (see p. 321).

3. *Congenital cystic degeneration of the kidneys.*—A considerable number of these curious cases have been published, and most of them have been collated by Virchow in two elaborate papers (Gesammelte Abhandlungen, pp. 837 and 864).

Kidneys in this condition present an enormous proportionate bulk, being as large as, or larger than, the kidneys of adults. In all but two cases both kidneys were affected. In several instances embryotomy was required to effect delivery, on account of the immense size of the abdomen. The fœtus (generally expelled prematurely) is necessarily still-born if both sides are affected, on account of the pushing up of the diaphragm, and the mechanical obstacle thus created to the expansion of the lungs.

Dr. Lever (Path. Soc. Trans. 1848-9, p. 74) has recorded the following typical example. The fœtus was one of eight months. It was club-footed and club-handed; it had six fingers on the left hand and as many toes on each foot. There was a hernia cerebri (? encephalocele) at the posterior part of the head. The thoracic and abdominal viscera were natural, except the kidneys. The right kidney weighed 4 oz. 6 drs.; the left, 4 oz. 1 dr.: they were irregular on their surface from numerous projecting cysts. On a section being made through the centre of each, it was found that all trace of kidney structure had disappeared, and that its place was occupied by an infinite quantity of cysts of different sizes, forming the whole mass of the organ; the calyces were in part normal, but large, and the pelvis of each kidney was perfect, with the exception that it formed a blind sac, with no opening; that is to say, there were no ureters. The bladder was small and empty; there was no trace of ureters on its external surface; but internally, at the spots where the ureters should have entered, there were small imperforate papillæ.

The structure of these kidneys was examined by Dr. Gull under the microscope. He could not detect any secreting tissue, and considered the cysts to be obstructed and dilated Malpighian capsules.

The degeneration has not always been found in so extreme a degree as in this case of Dr. Lever. Generally, some remnants of secreting texture (uriniferous tubes and Malpighian tufts) have been detected in the interstices between the cysts. In some cases the external surface is smooth, while the interior presents a spongy or cavernous structure, which, under the microscope, resolves itself into myriads of minute cysts. The researches of Virchow and Förster have fully demonstrated, that the cysts in these cases are originally produced by dilatation of short sections of the uriniferous tubes into pouches; these pouches afterwards become enlarged and separated from each other, and at length form distinct cysts. They are lined with a tesselated epithelium, and contain, at first, a urinous fluid, which, at a later period, when the cysts attain a larger size, becomes albuminous.

It is curious that malformations of the pelvis of the kidney, of the ureter, bladder, or urethra, or of some other part of the body, nearly always co-exist with congenital cystic degeneration of the kidneys. Sometimes, however, the lower urinary passages are perfectly open.

Virchow first pointed out the mechanical cause of this disease. In all the cases examined by him, there was found an imperforate state (atresia) of the straight ducts which terminate on the papillæ; and he conjectures that this had arisen from intra-uterine inflammation of the ducts of the papillæ, which ended in adhesion of their parietes and closure of their calibre. He further believes that the usual cause of this inflammation is the impaction of uric acid or the urates (Harnsäure-infarct) in the straight canals (see p. 402). The closure of the excretory ducts necessarily causes stagnation and accumulation of the urine throughout the entire organ, and leads to dilatations of the uriniferous tubes and Malpighian capsules, and the ultimate formation of cysts.

4. *General cystic degeneration of the kidneys in adults.*—This is a somewhat rare condition, though most museums contain specimens. There are two very fine examples in the collection of the Manchester Infirmary. In this form of disease the organs are greatly enlarged, so as sometimes to weigh several pounds, and to constitute tumours in the abdomen recognisable during life. Both kidneys are always affected; but not, generally, in an equal degree. The substance of the gland is converted into a mass of closely aggregated cysts, lodged in an abundant matrix of connective tissue (see Fig. 48). The cysts do not communicate with each other, nor with the calyces—except in rare cases, when some of them suppurate and open into the pelvis. They range in size from

a pin's-head to an orange, and have walls of varying thickness. Their contents also vary : some contain a limpid yellowish or reddish serum ; others a gelatinous substance. The fluid within the cysts always contains albumen, but not urinous ingredients. The interior of the cyst is lined with epithelium; and sometimes blood-discs, pus corpuscles, and cholesterine crystals are found within them. In far advanced cases the secreting tissue of the kidney is almost entirely destroyed ; more frequently remnants of renal tissue are found in the fibrous matrix between the cysts and in the pyramidal portions. The pelvis, ureter, and bladder are open, and usually healthy. Two or more cysts may become confluent by absorption of some parts of their walls, and then an irregular cavity is produced, with fibrous bands or fræna passing from side to side.

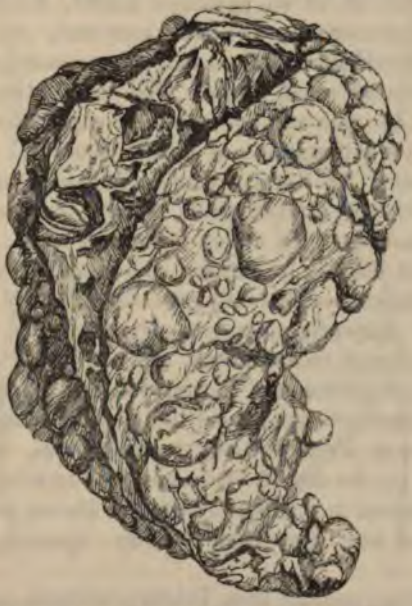

FIG. 48. General cystic degeneration of the kidney in an adult—from a preparation in the Museum of the Manchester Infirmary—one-fourth the actual size.

Queckett attributed the formation of these cysts to dilatations of the Malpighian capsules ; but the observations of Dr.

Conway Evans,* and Dr. Bristowe,† on what appear to have been incipient cases, lead to the conclusion that they are formed, as in the congenital cases, by expansion of sections of the uriniferous tubes, and occlusion and atrophy of the intermediate portions. Independent sacs are thus constituted, which at first are so minute that they can only be seen with the microscope, but at a later period they enlarge into visible cysts.

The clinical history of these cases has been but imperfectly studied. Of ten well-marked cases, which I have been able to collect, seven were men, and three women ; most of them were about the middle age ; six were between forty and fifty years of age ; one was "old," one was thirty-nine, and the youngest thirty. The symptoms during life are not very distinctive. The course of the disease is essentially chronic ; the secretion of urine goes on to an advanced period, without marked diminution—it may even be greatly increased. An unnaturally low density would appear to be a tolerably constant phenomenon, at least in the advanced stages. The end (if the patient die of the renal affection and not of some complication) is usually sudden, with manifestations of uræmic coma and convulsions. In a case cited by Rayer, there were recurrent attacks of excessively violent lumbar pains, severe gastric symptoms, abundant discharge of a watery urine, and lastly, convulsions, delirium, and coma. In another case recorded by the same author, the patient—whose only previous sufferings consisted in old-standing dyspeptic symptoms—was suddenly seized with coma, resolution of the members, and convulsive upturning of the eyes, which proved fatal in twelve hours. Albuminuria and recurrent hæmaturia are among the most constant symptoms. In Dr. Conway Evans's case, the urine was, however, not albuminous on the day of death, nor two months previously. Death was caused in this case by cardiac disease, and the renal degeneration was not, comparatively speaking, very far advanced.

The following example from Bright's memoirs on abdominal tumours (New Syd. Soc.'s publications, vol. vi., p. 208) shows the successive appearance of a renal tumour first on the left, then on the right side of the abdomen, and gives an excellent picture of the disease.

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* Path. Soc. Trans., vol. v., p. 183.

† Ibid., vol. ix , p. 309.

Mr. ——, about thirty, seen by Dr. Bright, November, 1835. His aspect bespoke a man labouring under some formidable chronic disease. He was evidently much emaciated and greatly enfeebled. He passed a moderate quantity of urine, which was acid, light-coloured, and albuminous. His present illness dated about two years back, at which period he had decided hæmaturia, which continued at intervals for some time. Since that, he had never considered himself in health; he had, however, pursued his usual occupation till lately, but for the last four months he had been more decidedly an invalid. A tumour was to be distinctly ascertained in the left lumbar space, where it appeared pretty firmly fixed (see Fig. 49). It might be fairly grasped by the hand so placed that the thumb was near the spine, and the finger advanced into the hypochondriac region. The history of the case, the state of the urine, and the situation of the tumour, all led to the easy decision that the tumour depended on enlarged kidney. When felt in front, the spleen, or the descending colon

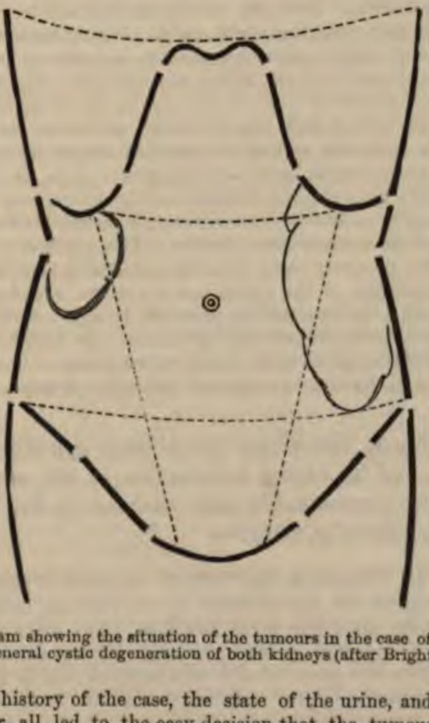

FIG. 49. Diagram showing the situation of the tumours in the case of a patient with general cystic degeneration of both kidneys (after Bright).

loaded with fæces, suggested themselves; but the fact that it seemed to belong rather to the posterior than the anterior part of the abdomen, and its fixed feel, would have removed these doubts, had not the history of the case pointed so distinctly to the kidney. The exact nature of the renal disease was less obvious. The very considerable enlargement of the organ did not belong to the usual history of albuminous urine, and the general loss of power bespoke some formidable organic disease. He was ordered a well-regulated nourishing diet. The Emplast. Ammoniaci c. Hydrarg. was applied to the seat of the tumour; and the uva ursi in infusion, and slight alkaline preparations, were directed to be taken. Under this treatment flattering reports were at first received, but the disease advanced, all the symptoms became worse, enlargement of the right kidney also became perceptible, the urine remained moderately coagulable (about a pint and a half in twenty-four hours), and he suffered a great deal of pain at the neck of the bladder, from the frequent passing of fibrinous coagula of a slight pinkish-yellow colour, about an inch long, and apparently moulded by the urethra. His emaciation became extreme, and he had frequent returns of hæmaturia. From the middle of February he was completely confined to his bed, expecting death daily. In the first week of April he experienced some slight convulsive seizures, and fell into a state of coma for a few hours before his death, which occurred about the 10th of April.

Both kidneys presented most extreme specimens of vesiculated disease; the left was the largest, and was probably eight or ten times the natural size, while the right was at least six times the size of the healthy kidney. The whole appeared made up of a congeries of vesicles, from the size of a pigeon's egg to a pea; and the substance of the kidney was almost obliterated; nothing but a thin layer of secreting structure remaining, and that greatly altered from the natural texture. The pelvis of the kidney and the mammillary processes alone retained a tolerably healthy appearance; the lining membrane of the pelvis had no undue vascularity, and was perfectly smooth; the mammillary processes, though somewhat flattened, showed, when divided, the healthy organization; the ureters were healthy, but the renal vessels, particularly the veins, were large. The other viscera were healthy, and the bladder contained half a pint of urine.

The enormous size which the kidneys sometimes attain in this disease, and the abrupt termination of life, are illustrated by the following remarkable case recorded by Dr. Hare (Path. Soc. Trans., 1850-1, p. 131):—

A man, æt. 46, was seen in Jan., 1850, for an attack of pleurisy. Under treatment, he recovered and returned to his business, until the 5th of March, when Dr. Hare was again called in. The night previously he had passed a considerable quantity of very bloody urine, which had, apparently, given him great relief from a constant pain he had in the left loin. On examination of the abdomen, which had lately become larger than usual, a tumour was found, extending from the cartilages of the false ribs of the left side to about an inch below the level of the umbilicus, and forwards to within about an inch of the median line. On percussion it was dull, and there was no interval of resonance between the tumour and the cartilages of the ribs; and the dullness on percussion extended upwards beyond the lower margin of the latter; the anterior border was rounded, but presented no signs of fluctuation.

In April, a considerable alteration was observed to have taken place in the tumour; it still felt solid and without fluctuation; the lower border extended an inch and a-half below the level of the anterior superior spine of the ilium. Its anterior border was deeply notched on a level with the umbilicus, and percussion was resonant at this notch, as also for some distance obliquely across the tumour. It presented very much the physical signs of a double tumour, or of two tumours; on placing one hand over the lower part of the abdomen, and pressing with the other against the left loin, although the tumour could be very slightly moved, it appeared to move as one mass. Dr. Bright, who also saw the case, spoke confidently as to its being "all kidney with intestine passing over it, and thus giving the appearance of two tumours;" there was also now a slight interval of resonance between the cartilages of the false ribs and the tumour. The urine, which had contained blood two or three times, was now clear.

On the 9th of December, he fell suddenly from his chair, convulsed and insensible; this was followed by sleepiness, numbness in both hands, and frequent twitchings.

On the 12th, when seen by Dr. Hare, he had a vacant expression, wandered a little, but answered sharply when spoken to; there were slight twitchings of the upper extremities. Pulse 72; feet and legs rather œdematous. The tumour appeared much the same as in April, except that it extended rather beyond the median line, and that the notch, at its anterior border, was less marked. Urine, pale, without sediment, sp. gr. 1008, containing one-tenth albumen.

On the 13th, he had two fits, somewhat similar to those on the 9th, and he died on the 16th, probably from the presence of urea in the blood.

Autopsy.—The left side of the abdomen was occupied by an enormous tumour, which proved to be the left kidney, the intestines being pushed over to the right side. The tumour also extended under the intestines half-way across the right half of the abdomen; its upper surface was adherent to the diaphragm, and it had so compressed the spleen, that the latter formed, as it were, a cap to the kidney; the pancreas was carried forwards, and was adherent transversely to the anterior surface of the tumour, near its upper part; the descending colon, somewhat contracted, was likewise adherent to its anterior surface, but perpendicularly, so as to divide it into two nearly equal portions.

The kidney measured 15¼ inches in length, 9½ in breadth, and about 23 in circumference, and weighed exactly 16lb.; it still retained somewhat the kidney shape, but its surface was uneven from the projection of different cysts. It consisted of one enormous congeries of cysts, varying in size from a small pea, to a cavity holding more than a pint of fluid; the larger cysts were at the surface, the smaller ones being about the centre; many of the smaller cysts projected more or less into the cavity of the larger ones; they presented different tints, from a dark purple, to a light straw-colour (the latter much more rare than the former), according to the colour of the contained fluids; the darker fluid was generally the thickest, and at the bottom of those cysts there was more or less of a dirty-red grumous-looking matter, which was wanting in those containing the lighter-coloured fluid. The thickness of their walls varied generally in proportion to their size, the larger having the thickest parietes. No trace of the proper structure of the kidney was discoverable. The fluid, under the microscope, showed an immense number of blood disks (more abundant in the darker fluids), some oil globules, exudative corpuscles, portions of the tubules of the kidney, and a considerable number of plates of cholesterine.

The right kidney presented incipient disease of the same kind, and was enlarged to double its natural size.

There was a slight hypertrophy of the heart, and a hernia above the umbilicus: the remaining viscera were natural.

In the following case, described by Dr. Gray (Path. Soc. Trans. vol. vi. p. 267), the disease appeared to be occasioned by external violence; death was not preceded by cerebral symptoms, but by vomiting and hiccough, possibly of uræmic origin:—

A man, æt. 40, much emaciated and anæmic, who had been very intemperate when young, was admitted into St. George's Hospital, March 7th, 1855.

He dated the commencement of his present illness seven years ago, when he received a severe injury of the back; for some time after this accident he passed blood in his urine. He then partially recovered, but during the three following winters he often passed a small quantity of blood, and suffered much from pain in his loins. Five weeks before his admission into the hospital, he fell on his right hip, and then the hæmaturia and pains in the loins returned in an aggravated form. When first admitted, he was in a very weak and exhausted condition; the pulse was exceedingly feeble, and the urine was loaded with blood; vomiting and hiccough supervened, and he died exhausted on the 10th of March.

Autopsy.—There was extensive cystic transformation of both kidneys; and to such an extent had the disease proceeded that the natural structure of the glands could in no part be detected. The right kidney weighed 3lb. 10oz.; the left, 3lb. They were each about 10 inches in length, lobulated on their surfaces, and composed of numerous separate cysts, varying in size from a pea to a small apple. The contents of some of the cysts were transparent and colourless, of others faint yellow, of others chocolate. The colour in these last appeared to depend on blood disks and their *débris*. The pelvis of the kidney and the ureter were not dilated.

All the other organs were natural, with the exception of the lungs, which were œdematous.

The primary lesion in this class of cases is probably the same as in the congenital cases, and consists in a progressive occlusion of the ducts of the pyramids, leading, at a later period, to saccular dilatations of the tubes of the cortex, and finally to the formation of myriads of separate cysts. The occlusion of the ducts of the pyramids may (conceivably) arise from inflammatory adhesion of their parietes, or from obstruction of their calibre by plugs of coagulated blood. In Dr. Gray's case, just related, the latter explanation would appear to be a not improbable one.

General cystic degeneration has evident affinities with the granular atrophic forms of Bright's disease, and probably requires a similar treatment.

CHAPTER X.

CANCER OF THE KIDNEY.

Wilson—Dis. of Urin. Organs. Lond. 1821, p. 234.
Otto—Neue Beobachtungen zur Anat. u. Path. Berlin, 1824.
König—Krankh. der Nieren. Leipz. 1826, p. 242.
Langstaff—Med. Chir. Trans., vol. viii., p. 294.
Bright—Memoirs on Abdominal Tumours. New Syd. Soc., vol. vi.
Rayer—Mal. des Reins, t. iii., p. 675.
Walshe—Nature and Treatment of Cancer. Lond. 1846.
Lebert—Traité d' Anat. Path. Paris, 1857, vol. ii.
Köhler—Krebs-und-Scheinkrebs-krankheiten. Stuttgart, 1853, p. 414.
Basham—On Dropsy, p. 339.
Döderlein—Inaug. Diss. Erlangen, 1860.
Rosenstein—Nierenkrankheiten, p. 397.
Kussmaul—Würzb. Med. Zeitschr., 1863, p. 24.
West—Diseases of Infancy and Childhood. Lond., 1852, p. 490.
Path. Soc. Trans., i., pp. 120, 281 ; vii., p. 268 ; viii., p. 286 ; x., p. 188 ; xiv., p. 179.
Edin. Med. Journ., xvi., p. 381 ; xix., p. 160 ; New Ser. i., p. 149.
Journ. f. Kinderkrankh., xxix., p. 396 ; xxxv., p. 426 ; xxxviii., p. 292.

Cancerous growths of the kidney may be *primary* or *secondary*. Primary cancer is attended by its proper symptoms and physical signs : it runs a distinctive course, and constitutes the cause of death. Secondary cancer, on the other hand, occasions neither symptoms nor physical signs : it is either a part-manifestation of a general cancerous cachexia, or an incident in the progress of primary cancer of some other organ ; and its existence is usually unsuspected until the autopsy. It is therefore necessary to consider the two conditions apart—the latter indeed very briefly, as it has little, if any, clinical interest.

A.—PRIMARY CANCER OF THE KIDNEY.

The following description is based on an analysis of 54 cases, of which 52 were collected from various sources, and two contributed by myself. In all of them the disease was followed to its fatal termination, and the diagnosis verified by dissection after death.

The cases naturally fall into two groups—*children* and *adults;* and it will be desirable occasionally to distinguish the one class from the other. The first group embraces 19 cases under the age of ten years—indeed all, except three, under four years. The second group includes 35 adults between the ages of nineteen and seventy.

Morbid Anatomy.—The species of cancer found in the kidney is almost invariably the encephaloid (fungus hæmatodes). It varies greatly in consistence and vascularity. In one instance it is described as being as soft as the milt of a fish ; more commonly it is about as hard as human brain. The mass is seldom of uniform consistence throughout, and it is frequently the site of extensive hæmorrhage. Cavities containing as much as a pint or more of clotted or fluid blood, or of blood mixed with cancerous detritus, have sometimes been found within the tumour.

Schirrus is very rare in the kidney. Wilson mentions such a condition, but his description is vague. Rayer in one instance found a mass resembling mammary schirrus in the midst of an encephaloid kidney : and Dr. Walshe, among the unpublished drawings of Carswell, discovered one of schirrus of the kidney : "the entire organ was converted into a gray-coloured substance, somewhat transparent, and of the hardness of fibrous tissue. It was intersected in various directions by pale-coloured bands which were opaque and firmer than the intermediate grey substance. It yielded only a small quantity of serosity on pressure, and presented few or no blood-vessels." (Walshe, l. c. 380.)

Colloid has been occasionally found forming a part of an encephaloid kidney.

Epithelioma has not, so far as I know, been found in the kidney, except in one case, published by Robin. In this, the right kidney of a man aged fifty-one was replaced by a large mass of adventitious tissue, of which part was soft and part hard. Both portions were composed of cells of epithelial character, most of them closely approaching the appearance of pavement epithelium, and attaining in the softer portions enormous dimensions. Some of the largest measured $\frac{1}{10}$ of a millimetre in length. None were found disposed in nests as in an ordinary cutaneous epithelioma.*

Encephaloid invades the kidney sometimes in the nodular, sometimes in the infiltrated, form. It always begins in the cortical substance, and afterwards involves the pyramids. The tunica propria is commonly thickened into a strong fibrous membrane.

When the whole organ is uniformly infiltrated, its natural shape and position may be tolerably preserved, even when it is enlarged to many times its original volume. But when a nodule grows from one end of the gland, leaving the remainder exempt, an irregular tumour is formed, which may assume shapes, and grow into situations very embarrassing for the diagnosis. The exempted portions rarely preserve their healthy state: the secreting structure wastes and degenerates; or it suppurates—though this is rare.

It is a marked characteristic of primary renal cancer, that it forms a tumour generally of large, often of gigantic proportions, which may stretch from the loin to the umbilicus, and from beneath the ribs to the pubes, and weigh many pounds. In 20 out of our 54 cases, exact information is given as to the weight of the tumour. In 10 children its average weight was 8¾ lb.; the smallest was 2¼ lb. and the largest 31 lb.! In 10 adults the average weight of the tumour was 9¾ lb.: in one case the growth was about the size and weight of the natural kidney; in two others it weighed 1½ lb.; in several it varied from 3 to 11 lb., and in one attained a weight of 27 lb. The enormous masses found in young children are really remarkable. In one example, recorded by Mr. Spencer Wells, a growth weighing between 16 and 17 lb. was taken from the body of a child only four years of age (Path. Soc. Trans. xiv. 179).

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* Ch. Robin. Memoir sur l'Epithelioma du Rein. Paris, 1855. See also Lebert, Anat. Pathol., ii., 351.

The surface of an encephaloid kidney is usually soft, irregularly lobulated, and of unequal consistence. Not unfrequently it yields a deceptive sense of fluctuation, especially in certain spots.

Renal encephaloid is liable to the same accidents (degeneration, softening, suppuration, hæmorrhage) as soft cancer elsewhere. In an instance mentioned by Bright, the softened mass burst into the peritoneum; in another, by Rayer, a cancer of the right kidney ulcerated into the duodenum; in a third, by Abele,* the disease broke through the abdominal parietes a fortnight before death, and formed a fungous ulcer through which a portion of the colon protruded and ultimately mortified.

The tumour generally contracts extensive adhesions to the surrounding parts. The colon is invariably found in front of the growth—though sometimes flattened and empty. The other abdominal viscera are thrust aside as the tumour enlarges: the intestines are pushed over into the opposite flank. When the growth affects the right kidney, the liver is displaced to left, often twisted on its transverse axis so that its upper surface takes a vertical direction and applies itself to the costo-abdominal wall. This distortion has been especially observed where, as in Döderlein's case, the growth protrudes from the upper end of the kidney, and makes its way into the right hypochondrium. When the tumour is constituted by the left kidney the stomach is pushed to right, and the spleen carried high up into the vault of the diaphragm. The thoracic viscera are displaced upwards more or less according to the bulk of the tumour, and in various directions according to the side affected. Among other effects on the adjacent parts, caries of the vertebræ was twice found: more or less compression of the inferior cava generally exists towards the later periods, occasioning œdema of the legs and sometimes (though rarely) ascites.

The pelvis of the kidney was found generally more or less involved, and, in the majority of cases, the ureter was permanently occluded by extension of the cancerous growth into it, or by blood-clots, or by the pressure of the main tumour.

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* Schmidt's Jahrb., Bd. v., p. 379.

The renal veins in several instances contained encephaloid matter; and in some of the cases it could be traced as far as the vena cava.

In the overwhelming majority of cases only one kidney was affected. Out of 53 instances which supply information on this point, the disease was confined to one kidney 47 times. In six cases both kidneys were involved; but in two only of these did the disease appear to be primary on both sides; in the other four one kidney was the seat of primary cancer, which formed a tumour, while its fellow contained small secondary nodules. Of the 47 unilateral cases, the right kidney was affected 27 times, and the left 20 times. The greater liability of the right kidney was nearly as marked in children as in adults.

The primary disease in the kidney was associated with secondary deposits elsewhere in 26 out of 42 cases which give details on this point: in the remaining 16 cases all other parts were exempt. The most frequent seat of secondary deposits were the lymphatic glands in the hilus of the kidney, and the vertebral and mesenteric glands. These glands formed in some of the cases a large tumour, which—as in a case to be presently related—transcended the dimensions of the renal tumour, and greatly embarrassed the diagnosis. The lungs and liver were also often affected; the other organs more rarely; but instances are on record in which almost every conceivable combination existed. The following table exhibits the distribution of the secondary deposits in the 26 cases already alluded to:—

Kidneys alone affected	16 cases.
Secondary deposits found elsewhere	26 ,,
Seat of secondary deposits :—	
Lumbar, mesenteric and vertebral glands	14 ,,
Lungs	13 ,,
Liver	11 ,,
Supra-renal capsules	4 ,,
Omentum	3 ,,
Heart	3 ,,
Vertebræ and rib	3 ,,
Bladder, uterus, penis—each	1 ,,

The infrequent association of primary renal cancer with cancerous deposits in the lower urinary passages, which this table shows, is somewhat remarkable, and is scarcely what one would expect, considering the close anatomical and functional relation of these parts.[*]

Etiology.—Renal cancer prevails at two distinct epochs of life—in early childhood, and in adult age. During adolescence the liability to it sinks to a minimum. Children under four years of age appear especially liable to renal cancer: 16 out of 53 cases occurred at this early period; three others between seven and ten years: the remainder were distributed almost equably between the ages of nineteen and seventy. The annexed table shows more exactly the relation of age to the frequency of renal cancer:—

	0—1 yr.	1—2 yrs.	2—3 yrs.	3—4 yrs.	7—8 yrs.	10 yrs.	
19 children .	1	4	6	5	2	1	
	19 yrs.	20—30 yrs.	30—40 yrs.	40—50 yrs.	50—60 yrs.	60—70 yrs.	Above 70 yrs.
29 adults[†] . .	1	6	4	3	7	7	1

The male sex is considerably more liable to renal cancer than the female. Fifty-two cases, in which the sex was distinguished, supplied 37 males and 15 females. The preponderance of the male sex is not so great in childhood as in adult age. Of 18 children, 11 were boys and 7 girls; of 34 adults, 26 were men and only 8 women.

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[*] While these pages were undergoing revision, I had an opportunity of examining a man (æt. 40) who died at the Royal Infirmary with extensive cancer of the stomach, combined with primary cancer of the right kidney. The latter organ was wholly converted into an encephaloid mass. I could not detect any traces of secreting structure in it: the mass was somewhat smaller than the natural kidney, and about the same shape. The left kidney was quite healthy, and greatly hypertrophied. The right ureter was pervious, but shrunk to about half its usual size. The left ureter was somewhat more capacious than natural. The right supra-renal capsule was wholly converted into a cancerous mass. There was very extensive cancerous disease (in nodules) of the liver, and of the vertebral glands.

[†] In six adults the exact age is not given.

The exciting cause of renal, as of other cancers, is wrapped in obscurity. In a few instances, a blow or a fall on the loins was the immediate precursor and supposed cause of the first symptom; but the disease had doubtless been already in existence before the accident, though concealed. In a case mentioned by Manzolini (Schmidt's Jahrb. B. 94, p. 74), a boy was kicked in the left side; this was followed by hæmaturia for fourteen days. Shortly after, a swelling appeared in the left loin, which eventually proved to be an encephaloid growth of the left kidney.

Symptoms and physical signs.—The distinctive symptoms of primary cancer of the kidney are:—*tumour in the abdomen* and *hæmaturia*. In every case in which the disease was the determining cause of death, one or both of these symptoms were present.*

Abdominal tumour is by far the most constant sign of renal cancer, and usually the earliest one noticed. Out of 52 cases there were only 2 in which a distinct intumescence could not be felt in the site of the kidney or thereabouts; and in both of these there was hæmaturia. In the remaining 50 cases a tumour was easily ascertained to exist in the abdomen; and in all but two it was of such size and prominence that it could not escape the most cursory examination. It is noteworthy that in *all* the children a large—nearly always an enormous—tumour existed.

The tumour presents itself first in the anterior lumbar region, between the margins of the ribs and the crista ilii; it then grows forward to the umbilicus, upwards into the hypochondrium, and downwards into the iliac and inguinal regions: in extreme cases it fills the entire belly. The tumour may, or may not, be covered with a ramification of enlarged superficial veins. The colon, and sometimes a portion of the small intestines, lie in front of it. This position of the colon furnishes an important diagnostic mark of all renal tumours. Percussion over the tumour is dull, except where the colon intervenes.[*]

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* Lebert states that he has known an instance in which the disease ran a latent course throughout: but he does not say whether in that instance the renal disease was really the cause of death.

To the hand the tumour feels smooth or irregularly lobulated, with rounded obtuse margins. The lobulations are often of unequal hardness, and a deceptive sense of fluctuation may be felt in places, or in the tumour generally. In Langstaff's case, a distinct and persistent pulsation was perceptible in the tumour; and a similar phenomenon was noted in Bristowe's case (Med. Times and Gaz. 1854, ii., 395). The fixity of the growth is usually a marked characteristic.[†]

The second symptom in importance is hæmaturia. Details on this point are supplied in 49 cases. Of these, 25 exhibited no trace of hæmaturia throughout their entire course. In 24 cases there was hæmaturia; but in 4 of these, there existed other possible causes for it than renal cancer (calculi, Bright's disease, external violence). These figures, even with this abatement, do not sufficiently express the danger of relying too strongly on hæmaturia as a sign of renal cancer. In 3 instances hæmaturia occurred only for a few weeks at the beginning of the complaint, and then altogether ceased—the urine thereafter continuing normal. In one case there was hæmaturia for a short period at first, and none during the remaining four years of life. In other cases hæmaturia did not appear until toward the last few months of life—perhaps years after the detection of a tumour in the loin. The absence of hæmaturia seems to depend generally on the occlusion of the ureter, either by the pressure of the tumour or the extension of the disease into it.

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[*] This position of the colon was not discovered in all the cases—generally, no doubt, from defective examination; but in some cases the detection of the gut may prove difficult, from its being compressed between the tumour and abdominal wall, and emptied of flatus. In doubtful cases, it might be of service to inject air per rectum, in order to inflate the collapsed gut.

The following remarks, by Bright, deserve to be borne in mind in searching for tumours of the kidney:—"In those diseases," he says, "in which it (the kidney) most rapidly increases, the enlargement shows itself much more towards the anterior part of the abdomen than towards the loins, not only because the firm structure of this part is more calculated to conceal a tumour, but also because, in the other direction, it meets with less immediate resistance; so that it often happens, while we are examining the lumbar region with the greatest care, and obtaining but a doubtful evidence of fulness and hardness by the eye and by the touch, and by careful comparison of the two sides, we can scarcely place the hand upon the anterior or even the lateral part without becoming at once sensible of the existence of a distinct tumour; and then, probably, by pressing that tumour backward, the other hand clearly informs us of its connection with the loins." (l. c. 199.)

[†] In the "Lancet" for March 18th, 1865, is an account of a case of moveable kidney affected with malignant disease. The tumour was mistaken for an ovarian growth, and operation for its removal commenced. The intestines were all *behind* the tumour.

When hæmaturia is present, it is a sign of very great value, and its character and features deserve attentive study. As a rule, it is irregularly intermittent and profuse. It recurs at intervals of a few days or weeks, usually without any appreciable cause. The tumour is not, of course, insensible to external violence: and in more than one instance a blow or fall on the loin has been the immediate precursor of the appearance of blood in the urine. In some cases the hæmorrhage is excessive, and followed by rapid anæmia and exhaustion, though this is rare. Generally the loss of blood is moderate, sometimes insignificant, and requiring the microscope for its detection. The formation of clots in the bladder, and their impaction in the urethra, is sometimes a source of severe suffering, and occasions excessive irritability of the bladder.

Other changes in the composition of the urine are sometimes found, but they are not distinctive. Of course, albumen always exists in the urine when it contains blood; more rarely albuminuria occurs independently of hæmaturia, from genuine Bright's disease, affecting either the exempted portions of the cancerous kidney, or the opposite organ. Not unfrequently, epithelial cells from the pelvis of the kidney and ureter are found in the urine, mixed with the blood.

The presence of cancer cells in the urine is a sign which usually figures prominently in the catalogue of symptoms of renal cancer, but its value is very doubtful. In all the later cases, especially where there was hæmaturia, the urine was carefully examined for cancer cells, but without success. Rosenstein mentions a case in which a cancerous villus was actually found projecting into the ureter, yet no cancer cells could be detected in the urine during life. It is by no means an easy matter to identify cancer cells in the urine, in consequence of their similarity to the transitional epithelium of the pelvis and ureter. It must be further remembered, that any cancer cells, which could find their way into the urine, must have escaped from parts of the growth which were broken down and degenerated; and to identify characteristic forms, in the ichorous detritus even of an external cancer, is more than I have ever succeeded in accomplishing: how much greater the difficulty, when that detritus has been further disintegrated by the action of the urine? In two examples of renal cancer, with hæmaturia, which I have had an opportunity of observing, repeated and careful examination of the urine failed to discover the presence of cancer cells.*

The other symptoms which have been noted in cases of renal cancer are less distinctive and constant than tumour in the abdomen and hæmaturia. The most important of them is pain in the hypochondrium and loin. This is sometimes an early symptom, and may show considerable severity. The pain is commonly intermittent; it shoots down in the course of the ureter to the inside of the thighs. It does not appear to be ever associated with retraction of the testicle. Pain is, however, wholly absent for long periods in a large number of cases; the tumour itself may be perfectly painless on handling, and give no inconvenience except from its weight and size.

Gastric symptoms—nausea, vomiting, anorexia—are common; and in several cases they were noted among the earliest symptoms. In other cases, again, none of these existed: the appetite was excellent; in four cases (all children) it was even voracious.

The general health varied exceedingly. In the majority of cases, rapid emaciation took place, going on at length to an extreme degree, with failing strength, and yellowish discoloration of the skin. In other cases, many months, and even years (in adults), passed over after the detection of the tumour, before the health seriously gave way.

The cancerous tint is not often mentioned in the list of symptoms, but this may have been from the brevity of many of the reports.

The bowels are generally disordered when the tumour attains a large size; diarrhœa, or obstinate constipation prevails; or the two conditions alternate.

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* Mr. Moore believes that he succeeded in identifying cancer cells in the urine drawn after death from the bladder of a man in whose kidneys cancerous nodules were found; but his description rather accords with the appearances of the epithelial cells which are always freely detached from the vesical mucous membrane after death. (Med. Chir. Trans., xxxv. 466.)

Towards the later periods, anasarca of the legs often sets in, and it may even extend over the whole body. Signs of constitutional irritation also present themselves, and become persistent. Life is at length worn out by gradual exhaustion of the vital powers: sometimes death is more suddenly induced by rupture of the tumour.

When there is no hæmaturia the urine is commonly normal; the healthy kidney becomes hypertrophied, and performs double duties. In no instance did uræmic symptoms arise.

The *duration* of the disease from the first appearance of symptoms to the fatal termination varied extremely. The duration was much shorter, as might have been anticipated, in children than in adults. Among the former, 14 cases are available for comparison: the mean duration was between seven and eight months; the minimum was ten weeks, and the maximum "over a year." In adults (20 cases available) the disease continued on an average two and a-half years; the extremes ranged from five months to seven years; 8 died under the twelvemonth, 7 under three years, 1 survived four years, 3 six years, and 1 seven years.

These numbers, as well as those having reference to the age of the patients, disagree with the statements current in books: and some of the numerous errors in the diagnosis of renal cancer may be traced to mistaken impressions as to the prevailing age and survivorship of patients so affected. The supposition of Walshe, indorsed by Lebert, that cancer of the kidney runs a more rapid course than other internal cancers, is not only unsupported by these larger numbers, but the contrary is clearly established, namely, that, as a rule, death is longer delayed in renal cancer than in primary cancer of any other internal organ.* The reason of this tolerance must be looked for in the duplication of the organ, the facility with which one kidney undergoes a compensating hypertrophy when its fellow is disabled, and takes upon it the work of the pair ; also the free room for enlargement which is afforded in the lumbar region, and the comparatively innocuous effects of displacement on the abdominal organs.

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* The mean duration of cancer of the pylorus, according to the combined statistics of Lebert, Herrich, and Popp, and Valleix, is under a year: out of 71 cases, 48 died within the year, and almost all the remainder (23 cases) within two years. The majority of hepatic cancers terminate probably under eight months—certainly under a year (see Köhler, pp. 308, 376). Walshe estimates the mean duration of cancer of the lungs at 13·2 months (l. c. p. 348); and he thinks cancer of the brain rarely lasts over a year (*Ibid.* p. 496).

The following examples will serve to illustrate the chief features of the disease :—

CASE I.—*Primary Cancer of the Right Kidney, and of the Lymphatic Glands in the Hilus. Secondary Cancer of the Liver, left Lung, and supra-renal Capsule.* (*From the notes of Dr. Renaud.*)

Hannah Hilton, æt. 59, a married woman, who had borne children, the last from eight to nine years ago, ceased to menstruate six years ago.

She first noticed a small and hard tumour in the right iliac space two years since, which made very little progress, and gave no pain or inconvenience for many months. She came under treatment in the early part of December, 1845, for a chronic diarrhœa, which had for some time past baffled all remedies. The evacuations were most copious, of a dirty olive colour, passed without pain, or accompanied with tormina. This ultimately yielded to the sulphate of copper.

I first examined the tumour about twelve months ago. It was painless, hard, and not bigger than a fœtal head, rising a little out of the right pelvic region. About a month from this, the tumour began to be painful and to increase. A feeling of crumpling parchment was noticed at its inner and lower portion. Shortly afterwards it began to extend upwards and backwards, in the direction of the loin ; there were flying pains also. The uterus, examined manually, was found free from the tumour, and apparently healthy. The colour of the skin was somewhat dirty, and this, together with the crumpling, were thought sufficiently suspicious to warrant a belief that the real nature of the disease was malignant degeneration of the right ovary.

I now lost sight of the case until three weeks prior to death, when I discovered that the tumour had gradually extended itself backwards, and that four weeks ago it began rapidly to grow, and spread in all directions, causing great pain and watchfulness. The woman emaciated very fast, was of a deep and dirty brown colour, had sunken eyes, and a look of suffering. The nature of the disease remained no longer doubtful, for the nodular portions of the fungus hæmatodes could be most distinctly felt beneath the abdominal walls. The feeling of crumpling was also more general, as also an occasional gurgling as of air, in the intestines. There had been no uterine hemorrhage, no difficulty or pain in passing urine, and nothing unusual in the character of the secretion. Opiates relieved the pain greatly. She died on February 17, 1847.

Autopsy.—Body greatly emaciated. In the abdomen there was a large fungoid tumour, extending quite across and to the right side and loins ;

passing obliquely over it was the colon, which was partially adherent, as were also some of the small intestinal folds. The tumour was not at all adherent to the interior of the abdominal walls. The uterus and ovaries were quite free from any disease, and were merely bound together with false membranous bands. The tumour had no pedicle, and though most carefully removed there were no connections found other than such as had been set up through peritoneal irritation. The abdominal cavity did not contain any dropsical effusion. The entire mass being removed, together with the liver, to which it was adherent, the right kidney was found so entirely degenerated into encephaloid matter, and so closely incorporated with the tumour, that nothing but a most careful dissection could have detected its true nature. It was enlarged to double the usual size, and no vestige of its proper structure remained; the vessels were however found entering the hilus, and the supra-renal capsule, also affected with encephaloid cancer, was in its usual position, and, in size and shape, bore a resemblance to a very large chesnut.

In the large tumour, which appeared to have its origin in the lymphatic glands of the hilus, were some cysts, filled with a grumous matter or with a semi-transparent jelly-like substance. The great mass was a homogenous and soft cancer, breaking down in most parts, but in some places yet as hard as cheese. The tumour was rounded, and about four inches thick, where it lay in the loins on the right side, and gradually became more thin toward the left margin, where it dipped beneath the stomach, surrounding the aorta and vena cava, in one portion of which cancerous matter was found. The extreme breadth of the tumour was nine inches.

The lower margin of the liver was cancerous where the tumour came in contact with it, and some other small cancerous tubera were found on its surface. The gall-bladder contained many calculi. On the surface of the lower lobe of the left lung were several tubera, and one as large as a small apple. The heart, left kidney, spleen, right lung, and other parts were healthy, and free from all traces of disease.

CASE II.—*Enormous malignant disease of the left kidney.* (*Lancet*, 1856, *I.* 626.)

J. B., aged six years, was admitted into the Middlesex Hospital, under Dr. Hawkins, May 29, 1855. J. B., born of healthy parents, was one of a family of ten children, of which five were still living, the others having died of acute infantile diseases. When the child was six weeks old his mother noticed that both left extremities were larger than the right; the skin was looser, and the muscles she describes as being less firm than those of the opposite side. She was so struck with the difference that she consulted a medical man about it. At three years of age the child had hooping-cough, and shortly after, measles, but he never had scarlet fever. The abdomen, the mother believes, was always rather larger than could be considered natural, but this had not been to a marked extent. With these exceptions, the child had fair health until the middle of April, 1855 (six weeks before admission), when he was suddenly seized with sickness; and from his appearance the mother believed him to be very ill, and though far better on the following day, so much so as to be able to walk out of the house, he did not regain his appetite for about a week. During this illness the mother accidentally discovered a tumour in the upper part of the left side of the abdomen. It then appeared to be almost circular, and about two inches in diameter. It was not perceptible to the eye, but its lower margin could be distinctly felt; it was very hard, but not painful, nor did moderate pressure cause any inconvenience. She believes that it gradually increased in size after she first discovered it, till she brought the child to the hospital, and during this time she noticed that he had quite regained his appetite, which had, in fact, become voracious, and though he appeared fatigued after moderate exercise, he was able to walk without effort.

State on admission.—Rather emaciated; abdomen very much swollen, especially on the left side, where the veins were enlarged and tortuous; the left extremities were considerably larger than the right, owing to the soft parts being much firmer and the muscles apparently better developed; there was, however, no difference in length. Upon manipulation, a tumour could be felt, of somewhat globular form, about three inches in diameter, occupying part of the left hypochondriac, left lumbar, and umbilical regions; its lower margin was well defined, but its upper boundary could not be ascertained—the dulness on percussion, which was complete over all parts of the tumour, being there continuous with that of the spleen. The patient ate, drank, and slept well, was able to sit up the greater part of the day, walked frequently up and down stairs, and did not complain of pain.

From this time the patient continued under observation until his death, a period of nearly twelve months. The tumour continued rapidly to grow, until it attained enormous proportions. On the 1st of August, the following note was taken:—"The tumour extends half an inch below the umbilicus, and about the same distance to the right of the mesian line; the abdomen generally is much more swollen, and the veins are much larger. The patient walks about the garden for an hour or two every day, and though taking a large quantity of food, and eating very frequently, is daily becoming more emaciated. He appears to suffer no inconvenience, except that caused by the bulk of the tumour, the large size of the abdomen being such as to impede progression. He complains of thirst, and evinces a desire to drink frequently of cold water. The bowels act with regularity; and the urine, which is frequently voided, and in quantity rather above the natural standard, presents no abnormal appearances."

The patient continued to go about till September, and to walk up and down one flight of stairs to and from the ward without assistance. About the middle of the month, after having spent some time in the garden of the hospital, he fancied himself unable to get back, and was then for the first time carried up-stairs. After this, he was almost constantly confined to his bed. The tumour gradually increased in size until his death; for some time previous to which, indistinct fluctuation could be felt in some parts of it. The abdomen, about the middle of December, measured in circumference 36 inches, and at the end of March upwards of 42 inches. For the last two months he suffered much from dyspnœa; and for the last three weeks, had constant orthopnœa, and daily increasing œdema of the left leg. The appetite, however, remained inordinate till the last; and the bowels, which had continued to act regularly till within a short time of his death, had recently become somewhat constipated. He sank gradually, and died April 7th, 1856.

The annexed admirable drawing of the patient was taken shortly before death, by Mr. J. Z. Laurence, and kindly placed by him at my disposal.

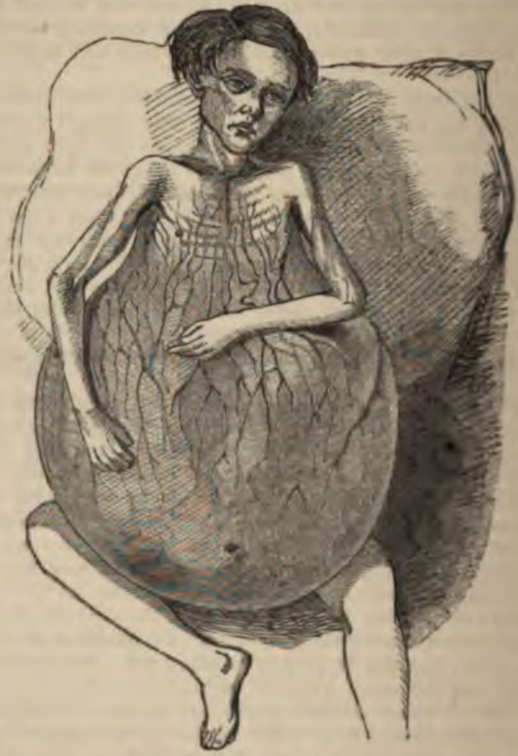

Fig. 50. Enormous cancer of the left kidney. Case of J. B. From a drawing by J. Z. Laurence.

Autopsy, fifty-four hours after death.—The whole of the abdomen, except the right inguinal region, was occupied by a large globular tumour, anteriorly firmly adherent to the parietes, and covered by peritoneum; posteriorly, lying in contact with the psoas muscle; the small intestines were thrust down to the right inguinal region; the spleen and liver were driven upwards into the thorax: the whole of the transverse colon was firmly adherent to the tumour; and a portion of the descending colon, which ran along the front, was for a short distance imbedded in it. The tumour, when removed from the body, weighed thirty-one pounds. Traces of kidney structure could be recognised, as if spread out over the entire substance; large masses of medullary cancer were visible on its surface. Upon section, the centre was found to be occupied by several pints of dark, thick fluid, floating in which were several fragments of the broken-down cancerous mass; the more solid portions varied in consistence from that of firm medullary cancer to gelatinous matter in a semi-fluid state, large masses of it being found in every stage of degeneration; the kidney on the opposite side was much enlarged. No cancerous deposit was found in any of the other viscera.*

Diagnosis.—We have seen that in nearly all cases of primary cancer of the kidney, a palpable tumour exists in the flank. If profuse hæmaturia co-exist with such a tumour, scarcely a doubt can remain as to the seat and nature of the disease.† But when there is no hæmaturia, the diagnosis becomes more difficult; indeed, there is scarcely any morbid condition which has been so frequently misapprehended. Renal cancer has been generally mistaken for enlargements of the surrounding organs—of the liver, spleen, ovary, or uterus; but sometimes for ascites, aneurism of the aorta, or perinephritic abscess. It has also been mistaken for tumours of the kidney of a different character—for pyonephrosis, hydatids, cystic degeneration, and hydronephrosis. Some of these errors were doubtless unavoidable; but most of them arose from an imperfect knowledge of the diagnostic marks of renal tumours, and from the undue weight attached to the absence of hæmaturia. As a positive sign, associated with abdominal tumour, hæmaturia—profuse, spontaneous, and recurrent—is of the highest significance; but its absence signifies comparatively little. In more than half the cases collected by me, hæmaturia was wholly absent from first to last; and in those cases in which hæmaturia was noted, intervals of many weeks or months elapsed in several of them, during which the urine was perfectly normal.

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* Some further particulars of the *post-mortem* appearances in this case are supplied by Dr. Van der Byl (Path. Soc. Trans. vol. viii.).

† The co-existence of these two symptoms is not, however, *absolutely* diagnostic of renal cancer. In a case of enormous enlargement of the spleen (leucocythæmic) recently in the Manchester Infirmary, there was profuse hæmaturia for several days. After death, some months subsequently, the kidneys and bladder were found perfectly healthy.

In those numerous cases, therefore, in which the observer derives no help from the examination of the urine, he must rely on his skill to ascertain the anatomical relations and nature of the abdominal tumour. In prosecuting this inquiry, he will especially endeavour to eliminate tumours of the liver, spleen, and ovaries—these being, from their comparative frequency, the most likely to lead astray.

If the intumescence occupy the right side, it may be distinguished from hepatic tumour, especially when not very large, by the possibility of tracing its upper limits below the margins of the ribs; the side of the hand can generally be so inserted at the edge of the ribs, that the tumour can be clearly felt to lie below it, and the liver above it. Along this line a coil of intestine usually lies, and yields a tympanitic sound on percussion. This sign is lost, however, when the renal growth contracts adhesions to the under surface of the liver; also when it projects disproportionately into the right hypochondrium, and displaces the right lobe of the liver. When this is the case, assistance may be obtained by feeling for the thin margin of the liver as it lies applied to the abdominal wall. Another important sign in such a case is the position of the colon. Hepatic tumours have no intestine in front of them (unless there be malposition of the viscera), and yield a dull note over their entire surface. Renal tumours, on the other hand, have the ascending colon in front, passing obliquely from below upwards and to the left; and the passage of flatus along the gut, or the clear percussion note over it, will rarely fail to detect its position.

A splenic enlargement is distinguished by the following signs:—absence of the descending colon in front; its rigid, somewhat thin, borders (not rounded); its extension upwards under the ribs; its mobility; generally, a tympanitic note is obtained in the extreme left lumbar region; often, on deep percussion, a bowel sound is perceived through its substance, which is not thick (a renal tumour is absolutely dull on the deepest percussion); antecedent history of ague or remittent fever, or evidence of leucocythæmia on examination of the blood; the direction of the enlargement is downwards and inwards to the epigastrium and umbilicus, and not toward the iliac fossa. It also rises higher toward the axilla than a renal growth. When the latter rises from the upper and fore part of the kidney, and pushes forwards and upwards rather than downwards, the diagnosis becomes very difficult, and depends mainly on the absence or presence of the colon in front of the enlargement, and hints derived from the previous history, or the state of blood on microscopic examination.

When the tumour presses forwards and downwards toward the umbilicus and the pubic and iliac regions, it is apt to be mistaken for ovarian tumour. The commemorative symptoms may here yield valuable information, though the statements of patients on such points are always to be accepted with reserve. An ovarian growth begins in the iliac fossa, and ascends; a renal growth begins in the flank between the ribs and the crest of the ilium, and descends. An ovarian tumour has no bowel in front of it, and the bowels are pushed into the lumbar region, where a clear sound can be elicited—exactly in the spot where the dulness is most complete when the tumour arises from the kidney. This last sign also serves to distinguish uterine from renal enlargements.

An encephaloid kidney can only be confounded with ascites when it is extremely soft, and fills the entire abdomen. The two conditions may be distinguished by the circumstance, that in ascites *both* flanks are dull, whereas in renal tumour one is dull and the other resonant.

When the tumour has been satisfactorily made out to be connected with the kidney, there still remain difficulties in deciding its nature. Malignant growths generally give a distinct impression of their solid structure. This distinguishes them from hydatid, purulent, and hydronephrotic cysts; but the consistence of the tumour is often very difficult to appreciate: if it be small and deep-seated, and the abdominal walls thick, the sense of fluctuation in a fluid cyst may be exceedingly obscure; on the other hand, encephaloid tumours sometimes yield a quasi-fluctuation which is very deceptive. In these doubtful cases, the presence of pus, or blood, or hydatids in the urine, of rigors, of nephritic colic, or of cancerous cachexia, supplies hints which incline the judgment in this or in that direction.

Prognosis.—The ultimate termination is, of course, always fatal. In judging of the probable survivorship of the subjects of renal cancer, the age of the patient is of great importance; the mean duration of the disease is at least three times as great in adults as in children. There is, however, nothing like exact proportion observed in this respect. In a girl of twenty-one, whose case is described by Langstaff, the disease lasted (with hæmaturia) for six years. Contrary to what might have been expected, the occurrence of hæmaturia does not appear to hasten the final catastrophe: the mean duration is almost exactly the same in the hæmorrhagic cases, as in those in which the urine was throughout normal.

The disease appears in some cases to become dormant for a while, making no appreciable progress for many months. In an instance of this kind recorded by Dr. Brinton, the stationary condition (which Dr. B. had flattered himself might pass into permanent obsolescence) came suddenly to an end, with death of the patient, through copious hæmorrhage into the tumour (Brit. Med. Journ., June 13th, 1857).

Treatment.—The management of a disease so hopeless is a melancholy duty. When the tumour is painless, and the urine natural, there is little for the practitioner to do beyond placing the patient in favourable hygienic circumstances. When the tumour is tender, or there are signs of local inflammation in its vicinity, warm baths or emollient applications may be used from time to time. It may be doubted whether it is prudent to interfere with a moderate hæmaturia. The losses of blood do not on the whole act disadvantageously. When, however, the hæmorrhage becomes excessive, means must be used to control it. Ice may be applied to the tumour, and acetate of lead or gallic acid administered internally. The clots which form in the ureter and bladder sometimes occasion the most poignant suffering by blocking-up the urethra, and causing retention of urine. The impacted masses should be pushed back into the bladder by means of a catheter, and the coagula broken up by washing out the organ with warm water.

As the disease advances, severe constitutional irritation sets in, which requires to be palliated by opiate and other anodyne medicines.*

B.—SECONDARY CANCER OF THE KIDNEY.

Secondary cancerous deposits occur in the kidneys, in the form of nodules varying from the size of a pea to that of a marble or walnut. Ten or twenty such nodules are not unfrequently found scattered through the cortical substance: the intervening renal tissue shows no sign of disease; the urine is normal, and no pain or other symptom betrays their presence during life. The following case offers an example, marked by some very unusual incidents, of extensive cancerous disease of the urinary organs, involving primarily the bladder and its vicinity, extending thence to both kidneys, of which the right was undergoing sacculation from compression of the corresponding ureter by the cancerous mass at the base of the bladder.

In January, 1862, I was requested by Dr. Crompton to see with him a shopkeeper, aged 38, who was then suffering from hæmaturia and paralysis of the bladder. The patient gave the following account of himself:—Three years previously, without known cause, he had an attack of hæmaturia, accompanied with excessively frequent micturition, pains in the back and bottom of the belly, but without vomiting or retraction of the testicle. These symptoms passed off, under medical treatment, in two months, and (apparently) complete recovery soon ensued.

After an interval of three years, during which the patient's health continued in every respect undisturbed, the present attack abruptly commenced. The patient was seized, six weeks before my visit, with violent pains in the loins and hypogastrium, accompanied by painful and excessively frequent micturition and bloody urine. All these symptoms came on simultaneously. There was neither sickness nor vomiting. The attempts to void urine were incessant—every ten or fifteen minutes during the day, and so constant at night that the patient scarcely obtained any sleep. Matters continued thus for three weeks; the patient, meanwhile, did not keep his bed, and he attended, as well he was able, to his duties in the shop.

But a new train of symptoms now showed themselves. The incessant micturition was succeeded by a total inability to empty the bladder, and the legs and belly began to swell rapidly. At this conjuncture Dr. Crompton's aid was obtained. On examining the patient he found considerable ascites, anasarca of the lower extremities, and retention of urine. Three pints of a sanguinolent urine were immediately withdrawn by catheter from the distended bladder; the patient was directed to keep his bed, and treated with alkaline diluents and nightly sedatives. Great relief followed this treatment, but the patient still continued unable to void a drop of urine spontaneously, and catheterism had to be practised twice a day.

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* A curious case is reported in the Philadelphia Medical and Surgical Reporter for 1861, p. 126. A man of 58 had had a tumour in the right hypochondrium for six years. It was supposed to be "cystic disease" of the liver; and his surgeons deliberately proceeded to remove it by operation. The tumour (which weighed 2½ lbs.) was accordingly removed, but on examination it proved to be the right kidney, wholly converted into an encephaloid mass. The patient survived fifteen days.

His condition at the date of my visit was as follows:—There was extreme pallor of the surface; considerable emaciation; no pyrexia; the tongue was moist, slightly furred. The legs were no longer œdematous, but considerable ascites still remained. The bladder was distended almost to the umbilicus; there was no pain, and the loins were not sensitive to pressure; nor was there any tumour to be felt in the renal region; the movements of the patient were active, and he was cheerful and lively.

About a quart of bloody urine was removed by catheter. A little pure blood came through the instrument first, then almost clear urine, and as the bladder became empty, the urine again became ruddy, the last few drops being almost pure blood. Dr. Crompton stated that a little bleeding always followed the morning and evening catheterism.

A careful examination of the urine yielded the following:—It was feebly alkaline from fixed alkali (derived from medicine); sp. grav. 1007; on standing, the blood-corpuscles subsided, and formed a very red, slightly clotted layer at the bottom of the urine-glass. Under the microscope there were found, in addition to the blood-disks, a few corpuscles with cleft nuclei —probably pale blood-corpuscles—but no renal elements—neither epithelium, nor casts, nor any suspicious (quasi-cancerous) cells of any sort, though diligently looked for. The proportion of albumen was no more than corresponded to the blood present.

The patient from this time gradually but steadily improved. The bladder slowly recovered the power to expel its contents; the urine became less and less bloody, and finally clear, and free from albumen.

Eight months afterwards (August 26, 1862) the patient waited on me. He was still pale and thin, but reported himself well, and had for the last six months been able to pursue his avocation.

I heard nothing more of the case until June 22, 1863, when I was summoned to visit the same man with Dr. Nesfield, under whose care the patient came after Dr. Crompton's departure from town. I found him in a desperate condition—emaciated to a skeleton; so weak that he could not turn in bed, nor raise his head from the pillow. There was no anasarca nor ascites. Great pain was complained of in the right renal region, but no tumour or fulness existed there. The urine was loaded with pus, and highly ammoniacal. Six days after, the patient died.

Autopsy.—On opening the abdomen and pushing aside the small intestines, a cancerous mass, half as large as the fist, was found implicating the base of the bladder, especially about the entrance of the right ureter. Within the viscus, a soft sprouting fungus, of the size of a hen's egg, was seen springing from the trigone; it was rounded in shape, elevated about an inch above the level of the mucous membrane, and very red. On and about it, occupying the inequalities of its surface, lay a quantity of calcareous or phosphatic matter, deposited in irregular masses. Small masses of a similar nature had been observed to come away with the urine for some weeks before death.

The right kidney was a little larger than natural; it felt flaccid and hollowed. On section, six cancerous nodules as large as marbles, and several smaller ones, were counted in the cortical substance. None of these were softened, nor communicated in any way with the pelvis of the kidney. The organ was sacculated to a considerable extent; the pyramids were in great part absorbed, and the remainder of the renal structure was converted into a reddish, leathery substance. The pelvis and infundibula were much dilated. The ureter was enlarged to the size of the index finger, and near its entrance into the bladder its calibre was almost effaced by the cancerous mass at the base of the bladder, through which it passed. Broken fragments of calcareous matter lay scattered in the dilated pelvis, which, together with the ureter, contained a quantity of urinous ammoniacal pus.

The left kidney contained eight or ten nodules similar to those in the right. The intervening renal tissue was perfectly healthy; the ureter was free, and the pelvis undilated.

This case presented several points of difficulty. At the time of my first visit the symptoms indicated pretty clearly an affection of the bladder: and as no stone could be detected on sounding, and no pus passed with the urine, the probability of the existence of a bleeding fungus seemed strong. The other possibility was renal calculus. The previous history favoured the latter view; the patient had recovered perfectly from his first attack of hæmaturia three years before—a result quite conformable with the idea of renal calculus, but much less so with that of fungus of the bladder. Then again, how explain the ascites and anasarca? They could not be attributed to the losses of blood and hydræmia consequent thereupon, for they passed away before the hæmaturia ceased. It appeared more likely, that the dropsical symptoms and the paresis of the bladder were companion phenomena, of a paralytic nature, produced by the reflex results of the antecedent intense irritability of the bladder, acting upon the nerves of the bladder and of the bloodvessels of the lower half of the body.

CHAPTER XI.

BENIGN GROWTHS IN THE KIDNEY.

Rayer—Mal. des Reins, iii. 605.
Godard—Substitution graisseuse du Rein. Paris, 1859.
Virchow—Gesammelte Abhandlungen, p. 208.
Heath—Adipose Transformation of the Kidney. Path. Soc. Trans. x. 199.
Dickinson—Fibro-fatty Tumour of the Kidney Path. Soc. Trans. xiv. 187.
Bristowe—A Tumour of the Kidney. *Ibid.* p. 189.
Wagner—Archiv der Heilkunde, 1860, Heft iv.
Friedreich—Archiv f. Path. Anat., Bd. xii.
Böttcher—*Ibid.* Bd. xiv.

In the records of medicine a number of cases may be found, in which the kidneys were the seat of adventitious growths of osseous, fibrous, fibro-fatty, cartilaginous, or glandular tissue. Generally speaking, such growths do not, unless they are large enough to constitute a palpable tumour in the abdomen, produce any appreciable symptoms during life; and they offer more of a pathological than clinical interest. They are all extremely rare.

1. *Osseous growths.*—Mention has already been made of the ossification which sometimes takes place in the fibrous septa which separate the compartments of a sacculated kidney (see p. 384).

Sometimes a fibrous or cartilaginous tumour grows in the substance of the kidney, and subsequently ossifies, transforming a large part of the organ into a bony mass. The tunica propria has also been known to undergo ossification. Rayer states that Dr. Elliotson sent to him two bony shells formed by the ossified tunica propria and pelvis of the kidney, taken from a man who died with symptoms of apoplexy.

2. *Fibrous and fibro-fatty growths.*—Dickinson and Bristowe have each recorded a case, in which the major part of the kidney was replaced by a morbid growth, composed of a matrix of fibrous tissue, in the interstices of which were soft masses of free fatty matter unenclosed in cells. In Dickinson's case the tumour weighed 6 lb. 7½ oz., and formed a perceptible tumour in the right hypochondrium. After death, a coil of intestine was found in front of the tumour, but so compressed and empty that its nature was not likely to be recognised during life.

In the case described by Godard, the lower half of the kidney was converted into a large mass of ordinary adipose tissue. A calculus of considerable size was lodged in the dilated pelvis. A somewhat similar transformation is described by Dr. Hullett Browne, complicated with calculous pyelitis, and renal fistula opening in the left loin (Path. Soc. Trans., xiii. 132).

Adipose tissue is, in other cases, deposited in great quantity, not in, but around the kidneys, so as evidently to interfere with their functions. In the museum of the Manchester School of Medicine there is a preparation in which a pale and atrophied kidney is enveloped in a firm investment of dense, granular, fibro-fatty tissue, fully an inch thick. The same tissue penetrates deeply into the hilus, so as to compress the bloodvessels and excretory channels.

3. *Lymphatic growths.*—Virchow, Friedreich, and Böttcher have described growths or deposits in the kidneys of leucocythæmic individuals, similar to those found under the same circumstances in the spleen and lymphatic glands.

4. Wagner has published two cases in which one kidney was converted into a large tumour, composed apparently of a combination of epithelial structure, fibrous tissue, and glandular (pancreatic) sarcoma. Both were female children—one nine months and the other eight years old.

CHAPTER XII.

TUBERCLE OF THE KIDNEY.

RAYER—Mal. d. Reins, t. iii. p. 618.
CARSWELL—Pathological Anatomy, pl. ii. fig. 5.
BASHAM—On Dropsy, p. 304.
ROSENSTEIN—Nierenkrankheiten, p. 384.
RILLIET AND BARTHEZ—Malad. des Enfans, t. iii. p. 852.
CHAMBERS—Decennium Pathologicum. Med. Times and Gaz. 1852, ii. 403.
SCHMIDTLEIN—Ueber die Diagnose d. Phthisis Tuberculosa der Harnwege. Deutsche Klinik, 1863.
KUSSMAUL—Beiträge zur Anat. u. Path. d. Harnapparats. Würzb. Med. Zeitsch., Bd. iv. p. 24.
MOSLER—Beiträge zur Path. u. Therap. d. Krankh. d. Harnwege. Archiv d. Heilkunde, 1863, p. 299.
COLIN—Néphrite Tuberculeuse aigue. Gaz. Hebd., t. x. p. 39.

DEPOSITS of tubercle in the kidney may be *primary* or *secondary*. In the former case, the kidney and its appendages are the seat of extensive disease, which runs on, attended with severe urinary symptoms, generally, if not always, to a fatal conclusion. In the latter, the deposits form a part-manifestation of general tuberculosis, or constitute incidents in the course of primary tubercle of the lungs, intestines, or some other organ; secondary deposits rarely give rise to symptoms, and are mostly unsuspected until the autopsy.

The comparative frequency of tubercle in the kidney may be judged of by the following numbers, which must be understood to embrace both primary and secondary deposits—the latter being, especially in children, by far the most frequent. Out of 1317 tuberculous subjects, examined in the Pathological Institution of Prague (out of a total of 6000 bodies), tubercle in the kidneys was found 74 times, or in the proportion of 5·6 per cent. of all tuberculous subjects.* Among 315 tuberculous children, Rilliet and Barthez found tubercle in the kidneys 49 times, or in the proportion of 15·7 per cent. From these statistics we may gather that the kidney is nearly three times more liable to deposits in tuberculous children than in tuberculous adults.†

A.—PRIMARY TUBERCLE OF THE KIDNEYS.

(*Tuberculous Pyelitis.*)

The statements made in the following pages are mainly based on an analysis of 31 cases, most of which are derived from the sources indicated at the head of the chapter.

Morbid anatomy.—The disease (which always implicates more or less extensively the excretory apparatus as well as the gland itself) begins in the kidney, and extends downwards into the pelvis, ureter, and bladder; or it begins in the pelvis, and spreads upwards into the kidney, and downwards towards the bladder; or all these parts may be invaded simultaneously or in quick succession. In the kidney, the deposit begins in the form of grey or yellow nodules in the cortical part: these afterwards coalesce into larger masses of crude tubercle, and extend into the pyramids. These masses at length soften in the centre, and eventually open into the infundibula. In this way abscess-like cavities arise, with anfractuous boundaries of tuberculous matter, which communicate with the pelvis, and discharge pus and broken masses of tubercle into the stream of urine.

In the pelvis and ureter, the deposit first begins in the submucous cellular tissue,‡ where it forms a rough, granular, semi-transparent or opaque layer. It subsequently softens and disintegrates, causing extensive destruction of the superjacent mucous membrane, which is discharged in shreds with the urine, mixed with pus and blood. The deposit is sometimes so abundant and uniform in the ureter, that that tube is converted into a thick rigid cylinder, of which the available bore is greatly narrowed, or even altogether obliterated. In a specimen submitted to me by Mr. Leach (case to be presently related), the interior of the pelvis was thickly incrusted with calcareous matter, and one of the ureters was completely occluded near its centre by an oval mass of tubercle about the size of a horse-bean.

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* Prager Vierteljahrsch., Bd. l. p. 1 (1856).

† I omit the statistics of Dr. Chambers, because there are some discrepancies in his tables which I have been unable to reconcile.

‡ See an observation by Dr. Handfield Jones, in the first vol. of the Path. Soc. Trans., p. 283.

Extensive destruction of the renal tissue eventually takes place, both from the encroachment of the tubercle-masses, and from sacculation and dilatation of the organ by the blocking up of the ureter (pyonephrosis). Sometimes no vestige of the secreting tissue remains; but more commonly certain portions are preserved, and these may present a moderately healthy appearance, or be far advanced in degeneration. In other cases the ureters are open and dilated, and admit free passage to the urine, pus, and tubercular débris; the kidney then maintains its normal dimensions, or it may even be contracted.

Actual tumour (pyonephrosis), detectable during life, is mentioned in 7 out of our 31 cases. It seldom reached great dimensions, but in one instance related by Ammon, it filled the entire side of the abdomen, from the false ribs to the crest of the ilium.

The disease is sometimes limited to one side, but much more frequently it invades both. Out of 28 cases which supply information on this point, the two sides were affected in 17, and one side alone in 11 cases. Of the latter, the right kidney was affected six times and the left five times.

In addition to the kidney itself, and its immediate appendages (pelvis and infundibula), the disease almost invariably involved the ureter (in 27 out of 28 cases), and very frequently the bladder (in 19 cases). The urethra was involved in six cases.

In the male sex, the disease not unfrequently implicates the generative organs (prostate eight times, vesiculæ seminales five times, testicles three times); but it is otherwise in the female sex. In only one instance (to be presently related) out of nine females, were any of the generative organs involved.*

The disease very rarely runs its entire course without the occurrence of tuberculous deposits in other and unconnected parts of the body. Twenty-six cases were examined with sufficient minuteness to supply information on this point. The lungs were affected 25 times; the abdominal glands, 13 times; the intestines, 18 times; the osseous system, 5 times; the peritoneum, 4 times; the spleen, 3 times; and the liver, once.

In one case the ulceration (tuberculous) in the bladder opened a communication with the rectum (Basham); in another, a vesico-vaginal fistula resulted from a similar cause (Mosler); in a third, the suppurated kidney burst into the peritoneal sac (Lundberg, Schmidt's Jahrb., Bd. xci. p. 74).

Etiology.—The direct exciting cause of renal tubercle is generally inscrutable. Cold is the cause most frequently mentioned; the patients came, in several instances, from conspicuously tuberculous families. Men are more liable to this complaint than women—in the proportion of 18 of the former to 11 of the latter. No age is altogether exempt. The youngest case noted was a child of three years and a half, and the oldest (mentioned by Dittrich, and not included in the table) was a man of seventy-one; but the greater number occurred in the middle periods of life. The following table gives the precise ages in 28 cases:—

From 0 to 10 years	3 cases.
" 10 " 20 "	4 "
" 20 " 30 "	5 "
" 30 " 40 "	9 "
" 40 " 50 "	3 "
" 50 " 60 "	2 "

Symptoms.—The symptoms are mainly those of chronic pyelitis, conjoined, in a considerable majority of the cases, with those of chronic cystitis. The complaint begins with a dull pain in one or both lumbar regions, accompanied with frequent micturition. At the same time the urine becomes turbid, and sometimes mixed with blood. When the disease is fully established, the urine is charged with a large quantity of pus, which forms a thick, yellowish layer at the bottom of the vessel. Blood is also usually present, either in microscopic quantity, or sufficiently to tinge the urine. The hæmaturia is, however, never profuse; in several instances it was noted that small, thready clots of blood were passed. Under the microscope, there are found, in addition to the pus- and blood-corpuscles, a number of oval and irregularly tailed cells from the bladder and upper urinary passages, together with granular débris, broken masses of softened tubercle, shreds of connective-tissue and elastic fibres.

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* The mutual independence of tuberculosis of the urinary and generative systems in the female, is further shown in a converse manner by Dittrich. Out of 45 cases of tuberculosis of the female genital organs, he only found one in which the disease also implicated the urinary organs (Archiv der Heilkunde, 1863, p. 304). Virchow describes an additional example of this rare conjunction, in which urinary tuberculosis was associated with secondary deposits in the vagina (Archiv für Path. Anat., Bd. v. p. 405).

The reaction of the urine is feebly acid. Very few exceptions to this rule exist, and those are due to ammoniacal decomposition of the urine from detention in some part of its course, as in Mosler's case, from the tumid state of the external genitals. The urine is necessarily albuminous from the presence of pus, but usually only in a slight degree. Casts of tubes are only mentioned once. Micturition is always excessively frequent; often dolorous. In one of Basham's cases, temporary alleviation of the pains followed each micturition: this has not been observed in other cases.

As the disease advances, great emaciation takes place, accompanied with hectic fever, sometimes marked by chills and rigors of tolerably regular recurrence. Persistent pains are felt in the back, in the lower part of the abdomen, and often along the urethra.

When the kidney is sacculated and enlarged, so as to form a tumour in the flank, the swelling is usually painful; it may, or may not, yield distinct fluctuation. Sometimes the tumour displays variations in its size: it enlarges when the ureter is dammed-up by the discharged débris, and becomes more painful, at the same time the quantity of pus in the urine diminishes—or, if the stoppage be complete, temporarily disappears. Anon the course of the pus and urine is re-established, and the tumour subsides and becomes less painful.

In the progress of the case, or towards its termination, the lungs and intestines generally betray the advance of tuberculous disease. Cough and oppression of the chest, or incontrollable diarrhœa, make their appearance. Gastric symptoms (nausea, vomiting, hiccough) are unusual; but in some cases, as in the two about to be related, they are a marked feature of the complaint. The absence of intestinal tuberculosis, accompanied with obstinate constipation, appears to favour their occurrence.

If both kidneys are affected, the extensive destruction of secreting tissue is liable to give rise to uræmic phenomena. The quantity of the urine is usually below the average; but exceptionally, as in a case recorded by Dr. Risdon Bennett (Path. Soc. Trans. VIII. 284), the urine is abundant and of low specific gravity. Usually death occurs from the exhaustive effects of the protracted and profuse suppuration, or from the severity of the pulmonary or intestinal complications.

The two following cases will serve as illustrations of the course of the disorder and of the general appearances found after death. The first occurred in the practice of my friend and former pupil, Mr. Leach, who kindly furnished me with the notes of the case, and the anatomical preparations. The second is from the clinique of Prof. Kussmaul, where the case was carefully watched for a period of six months.

CASE I.—*Tubercle in both kidneys, ureters, bladder, and urethra; in the prostate gland and vesiculæ seminales; also in the lungs and mesenteric glands.*

W. P., æt. 53, a brewer, had been ailing three years. His disease began with pain and difficulty in micturition. The urine was thick, and sometimes mixed with blood: though he made water very frequently, he did not think that he pased an excessive quantity. Except for short intervals, he had suffered from the same symptoms for the last three years. His urine had been occasionally quite clear, but generally thick, and often dark. He had never complained of much pain in the lumbar region.

About nine months before his death, the patient began to vomit frequently, especially after taking food: for the last five months vomiting after meals had been constant, frequently accompanied with pain in the epigastrium.

The general health had gradually failed during the last three years; but he lost flesh and strength more rapidly during the last twelve months. He worked occasionally, however, up to six months before his death. For many months he had felt a gnawing pain just over the pubes, increased by pressure: this was less severe during the last six months of life.

Five months before his death the edges of the meatus urinarius began to ulcerate, and the ulceration gradually widened the orifice to double its natural size. In the course of the last six months two small abscesses formed in the scrotum, both of which were opened, and subsequently healed.

He had been in the habit of taking large quantities of beer, but not much spirits: he contracted gonorrhœa many years ago, but he never had any venereal sores.

About a week before his death, he was in the following condition:—Emaciation very great; countenance sallow; meatus urinarius much enlarged and ulcerated; severe pain is felt along the urethra and in the glans penis, especially after voiding urine. The ulceration can be seen to extend for a depth of nearly half an inch into the urethra. The urine contains abundance of pus and a small quantity of albumen; no casts were found. The urine is passed very frequently, and in small quantities. He vomits after everything he takes, even after simple water, or a little brandy-and-water. What he brings up is a brownish liquid; it contains no sarcinæ; sometimes a little blood comes up; but he thinks it is derived from the back part of the nose, where he feels pain and rawness. The abdomen is flat, or rather depressed: the epigastrium is very painful on pressure. In the right hypochondrium a little hard mass can be felt on deep palpation, and there is dulness at this spot on deep percussion. There is also considerable pain on pressure in the hypogastric region. He complains of aching pain over the lower ribs on both sides. The bowels are very constipated, and have been so for some time.

The day before his death he vomited a considerable quantity of blood. The sickness and vomiting were somewhat relieved for a short time by effervescing draughts, with morphia, but only for a day or two. Afterwards the vomiting became continuous: he vomited, or attempted to vomit, every half hour or so. There was no delirium till the day before his death, which took place on the 27th of December, 1864.

Autopsy.—*Stomach* of normal size; mucous membrane congested in parts; no thickening of, or deposit in, the walls; pyloric valve thickened and somewhat contracted. *Liver* healthy. *Mesenteric glands* much enlarged; some of them contained small cretaceous masses. *Lungs*: *left* contracted and full of miliary tubercles; *right* contained hard masses of tubercle at apex.

Kidneys—*left* of natural size; on the outside, white, slightly raised spots are seen through the fibrous covering. On removing the latter, the surface of the cortex is seen marked with small white nodules, some of which are collected into patches; to these patches the tunica propria is tightly adherent. On section, several large cavities are opened into, containing pus. The largest of these is situated in the upper part of the kidney, and is lined by a smooth membrane, except at its opening into the pelvis, where some calcareous matter is deposited. All the other cavities open into the pelvis; some of them have irregular anfractuous boundaries of softening tubercle: these likewise are more or less completely lined with calcareous matter, composed of carbonate and phosphate of lime. The whole of the pelvis is encrusted with the same earthy material, which can also be followed for some distance down the ureter. In the cortical and pyramidal parts of the kidney intervening between the cavities, the renal tissue is studded with soft nodules of disintegrating tubercle, varying in size from a pin's head to a pea. The submucous tissue of the pelvis and ureter is the seat of a thick granular layer of grey tuberculous matter, softened in parts; the ureter is thereby converted into a thick, rigid, uneven tube, with a narrowed calibre.

The *right kidney* is much smaller than the left. The pyramids are occupied by abscess-like cavities full of pus. The septa between the pyramids are in some places preserved, in others partially broken down. The pelvis is greatly contracted, almost obliterated: in one or two places there are narrow communications between the sacs of pus in the pyramids and the unobliterated parts of the pelvis. The ureter is completely occluded, midway between the kidney and the bladder, by an oval nodule of yellow crude tubercle about the size of a horse-bean.

The whole of the mucous membrane of the *bladder* is strewed with deposits of tubercle. These are sparsely scattered and scanty, except over the trigone; here the deposit is very abundant, in the form of small granulations rather larger than a pin's head. A few similar granulations are seen in the prostatic part of the urethra.

The *prostate* gland is somewhat enlarged on the under surface, and contains two small tuberculous nodules. One of the *vesiculæ seminales* also contains softened tuberculous matter.

CASE II.—*Tubercle of the left kidney, pelvis, and ureter; of the bladder and urethra; also of the pericardium, lungs, peritoneum, and mesenteric glands.*

A needlewoman, 33 years of age, strongly built, whose father seems to have died of phthisis, took cold in consequence of a severe wetting in the autumn of 1859, about a year before her death. Her first symptoms were those of cystitis, with moderate fever. The fever soon disappeared; but the pain in the bladder, which radiated upward into the left loin, and the urgency and burning pain of micturition, together with a turbid condition of the urine, remained, and persisted through the winter. Impairment of digestion and emaciation were also observed.

About half a year from the commencement of her complaint, feverishness returned: pain and urgency of micturition increased, and blood appeared in the urine. From this time (February, 1860) the patient became the object of exact observation. She was already markedly emaciated, pale, with a hectic flush on each cheek; she suffered from headache, often from palpitation; the appetite was bad, the bowels confined, and there was moderate fever: the desire to pass water was constant, and the pain in the bladder, shooting into the left loin, great. The urine was scanty, tinged with blood, with a thick deposit of pus and blood-clots. The urethra was somewhat swollen and tender; and after micturition the bladder still

contained several ounces of unevacuated urine. These symptom
tained themselves without essential change for six months, until s
on the 4th of September.

The loss of flesh continued without interruption, and reached an
degree. Hectic fever prevailed, with evening exacerbations—the
rature rising to 38·5—39·5 C., and sometimes to 40 C. From the
March, severe paroxysms of chills and rigors, followed by heat and
ing, occurred at irregular intervals. At the end of July, night swe
bed-sores were noted.

Gastric symptoms were throughout prominent. They increas
diminished. For days together the patient would suffer from
epigastric pains, nausea, vomiting, and disgust of food. From the
May, she occasionally suffered from paroxysms of hiccough, lasting
hours. In the last months of life she was troubled with bilious vo
and towards the end she had diarrhœa.

The quantity of urine was invariably scanty, though the patient
times drank a good deal. The proportion of blood in the urine gr
diminished from the beginning of March, and the blood-clots son
were absent for several days, yet the blood never disappeared alto
The quantity of pus increased. From May onward there appeare
sionally in the urine sloughy shreds of cellular tissue, elastic
swollen bladder-epithelium, and little yellow broken masses of d
On the 8th of July, some epithelial renal casts were for the fir
discovered in the urine.

At the end of April a vaginal examination revealed the existen
small hard swelling at the base of the bladder (this was proved
autopsy to be due to a tuberculous thickening at the point of entr
the left ureter).

The patient began to complain of pains in the chest soon af
admission into hospital; then a dry cough came on, and later on,
dulness on percussion was perceived in the left infra-clavicular regi
the later periods she also complained of oppression in the chest;
never expectorated, and there never existed any of the more open sy
of pulmonary tuberculosis.

It was a singular and inexplicable circumstance, that a short tim
death an improvement took place, which lasted several days; all th
and the fever disappeared, the appetite returned, and the strength
far restored that she was able, unassisted, to sit up, although be
was scarcely able to turn in bed.

Autopsy.—Emaciation had reached the most extreme degree. Th
and its membranes were healthy. Some tuberculous granulatio
found in the otherwise healthy *pericardium* at the base of th
auricle. *Heart* healthy. The *lungs* and *pleuræ* were studded wi
and yellow granulations. The left apex contained several grey no
large as walnuts. The *liver* contained no tubercle, but its perito
vestment was thickly covered with grey granulations; the org
adherent, by its convex surface, to the abdominal wall. A mass o
culous glands, as large as a pigeon's egg, occupied the portal fissu

The *peritoneum* covering the spleen, intestines and mesent thickly covered with tuberculous granulations. The *mucous membrane of the intestinal canal* was throughout free from tubercle.

The *right kidney* was itself healthy, together with its pelvis and ureter; but its capsule was studded with miliary tubercles.

The *left kidney* presented its usual form and size, but its capsule was converted into a thick membranous covering which enclosed the degenerated gland. In the upper half of the organ were found one large and two smaller cavities, separated incompletely from each other by undestroyed renal tissue. These cavities were filled with a greenish-yellow muco-purulent fluid, and their anfractuous walls were composed of reddish and yellow cheesy deposit. The reddish and yellow materials, in the form of smaller and larger nodules, were deposited in close contact with each other. These cavities stood in direct continuation with the mucous membrane of the pelvis, which was similarly degenerated and thickened. In the lower half of the organ was another and a larger cavity, similarly constituted with the others, and, like them, communicating with the pelvis.

The undestroyed portions of the kidney had a pale red colour, and contained in the lower parts of the gland several small, roundish, greyish and yellowish cheesy nodules. The coats of the left ureter were several lines thick, firm and rigid; the mucous membrane tumid, friable, yellowish, and the seat of numberless miliary tubercles; in many places the ureter was superficially eroded into roundish ulcers, with firm raised edges.

The walls of the *bladder* were three lines thick; its cavity was contracted; its inner surface reddened, and riddled with ulcers having tuberculous margins. The peritoneal surface of the viscus was studded with tubercles. The serous coverings of the generative organs were in the same condition, but the organs themselves, with the exception of the ovaries, were healthy. The ovaries contained several grey tuberculous nodules. The lumbar glands contained cheesy matter.

The *duration* of the disease varies from a few months to two or even three years. Only 11 out of our 31 cases supply moderately exact information on this point. Four died under six months; three in six to twelve months; three in one to two years; and one survived three years.

The *diagnosis* of tubercle in the kidney and its appendages turns mainly on the existence of signs of chronic pyelitis, joined with collateral evidence of tuberculosis, and the absence of any other assignable cause of pyelitis (calculi hydatids, &c.). Examination of the urine furnishes important information; not only is the urine abundantly purulent, but it also contains a quantity of granular *débris*, sometimes mixed with broken masses of tuberculous matter (insoluble in acetic acid), shreds of connective tissue, and beautiful meshes of elastic fibres

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and other tonics, combined with a nutritious diet and a moderate allowance of stimulants. Opiates are generally required to ensure rest and some alleviation of pain. These means may be supplemented by the occasional use of the warm bath. To check excessive secretion of pus, the muriated tincture of iron may be given in doses of 15 or 20 drops thrice a day (see TREATMENT OF CHRONIC PYELITIS, p. 397).

B.—SECONDARY TUBERCLE OF THE KIDNEYS.

Secondary tubercle is deposited in the kidneys in the form of minute yellowish nodules and granulations, varying in size from a pin's head to a pea. The little masses are scattered over the surface and through the interior of the gland, chiefly in the cortical part. In places they run together into groups or patches as large as a sixpence or a shilling. The intermediate parts of the kidney are either altogether healthy, or only show signs of congestion immediately around the deposits. When the pyramids are affected, the little granulations sometimes evince a disposition to assume a linear arrangement parallel with the straight ducts. Such deposits are not uncommon in acute general tuberculosis; much less frequent in persons who have died from pulmonary or intestinal tubercle. The deposits are generally confined to the substance of the kidney, without participation of the pelvis and ureter.

Secondary tubercle is greatly more common in the kidneys than primary. Out of 91 cases of renal tubercle tabulated by Dr. Chambers, 76 were secondary, and 15 primary. *Both* kidneys are nearly always implicated in the former.

As a rule, no symptoms referrible to the kidney are observed during life. The urine presents merely febrile characters, and contains neither pus nor blood. If, however, the deposit take place with excessive rapidity, pains in the back and other indications of renal disturbance may occur. In the following case, by Colin, deposition of tubercle in the kidneys, occurring in the course of chronic phthisis, was thus diagnosticated during life.

A soldier, aged twenty, suffering under chronic phthisis, was suddenly seized with violent lumbar pains accompanied with an intense rigor. Next day, these pains were so violent as to cause the patient to cry out; the lumbar muscles were in a state of strong contraction and exquisitely tender. There was high fever, with a corresponding state of the urine. Three days later, acute meningitis set-in, which destroyed the patient in four days. The autopsy revealed exudation of lymph (but no tubercle) on the meninges; old pulmonary mischief, with recent deposit of miliary granulations in the lungs; the spleen was studded with similar granulations. The kidneys were markedly enlarged, the capsule easily detached; about thirty yellow nodules, as large as pins' heads, were scattered on their surface. On the convex border of each kidney there existed, in perfect symmetry, two whitish patches about the size of a two-franc piece, composed of an aggregation of a large number of granulations identical with the preceding. Sections of the organs revealed an immense number of similar granulations scattered in the cortical substance, and to a less degree in the pyramidal portion. It was calculated that each kidney contained from 300 to 400 of these granulations.—(Gaz. Hebd. x. p. 39.)

CHAPTER XIII.

ENTOZOA IN THE KIDNEYS.

The parasitic worms which infest the kidneys are : *Echinococcus hominis* or *hydatid*, *Bilharzia hæmatobia*, *Pentastoma denticulatum*, and *Strongylus gigas*. The first-named is by far the most common in these latitudes ; the second is the most common in Egypt, Cape of Good Hope, and certain other hot countries ; the two last are of extreme rarity. Sometimes intestinal worms wander into the kidneys and urinary passages (*erratic worms*) ; and in some notable instances, objects which were not parasites at all, or which were parasites wholly foreign to the human body, have been described and figured as genuine parasites of the urinary organs (*spurious worms*).

1.—HYDATIDS IN THE KIDNEY.

(*Echinococcus hominis.*)

Chopart—Traité des Malad. des Voies Urinaires. Ed. by Ségalas. Paris, 1855.
Rayer—Malad. des Reins. T. iii., p. 545.
Barker—On Cystic Entozoa in the Human Kidney. Lond., 1856.
Davaine—Traité des Entozoaires. Paris, 1860, p. 524.
Gervais and Van Beneden—Zoologie Médicale. Tom. ii., 274.
Béraud—Hydatides des Reins. Paris Thesis, 1861.
Curling—Med. Times and Gaz. 1863. ii. 164.
Collection of cases in Med. Times and Gaz., Feb. 17, 1855.
Sieveking—Lancet, Sept. 10, 1853.
Simon—*Ibid.*, Sept. 24, 1853.
Durand—Assoc. Medical Journal, March, 1851.
Tomowitz—Schmidt's Jahrb. Bd. 116, p. 200.
Quinquerez—*Ibid.*
Meissner—Beiträge zur Lehre von dem Vorkommen des Echinococcus beim Menschen. Schmidt's Jahrb. Bd. 116, p. 183.
Adams (Dr. Leith)—Lancet, Oct. 1, 1864.
Cobbold—Entozoa. Lond., 1864, p. 273.

Hydatids in the kidneys are comparatively rare; they are much less common than hydatids in the liver and even in the lungs; but they are more frequent than hydatids in the other organs and tissues of the body.*

Natural History.—A hydatid tumour consists of an adventitious *outer capsule*, composed of fibrous tissue, which is organically connected with the texture of the organ in which it is situated. Within this, and unconnected with it except by contact, lies the *hydatid cyst* itself. This latter varies in size from a walnut to an adult's head. The cyst-wall varies in thickness, according to the size of the cyst, from about a line to a tenth of a line or less, and is composed of an opalescent tremulous substance resembling boiled white of egg. When examined more closely, it is found to have a laminated structure (Fig. 51), and to be composed of an immense number of thin lamellæ or layers, which, under the microscope, exhibit a perfectly homogeneous structure. Within the cavity of the cyst a number of secondary or daughter-cysts float freely in a watery saline fluid, which is devoid of albumen. The daughter-cysts vary in size from an orange to a pea or pin's head: they may even be much smaller than this, and require a microscope for their detection. A mother-cyst may however be barren: that is, contain only fluid contents; but this is rare. More commonly twenty, thirty, a hundred, or even many thousand secondary cysts float within it. The structure and attributes of the secondary cysts are identical, in every respect, with those of the parent; and their walls display the same characteristic lamination.

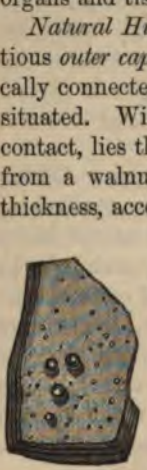

Fig. 51. Wall of a hydatid cyst, showing the laminated structure—not magnified. (After Davaine.)

Sometimes this constitutes the entire anatomy of a hydatid cyst; but as a general rule additional structures are found,

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* Davaine gives the following rough approximations of the relative frequency of hydatids in the different organs and tissues :—

Liver	166	Osseous system	17
Lungs	40	Parietes of the body	12
Kidneys	30	Heart	10
Pelvis	26	Orbit	9
Brain	20		

which indicate a more advanced phase of development: these are—a *germinal membrane* lining the interior of the cyst, and certain minute animalcules growing therefrom which are termed *echinococci* (*scolices* or *tænia-heads*).

The *germinal membrane* is a thin, transparent, homogeneous, (unlaminated) tough membrane, which forms an interior sac closely applied to the inside of the hydatid vesicle. When detached and emptied it shows a tendency to contract and curl on itself in a peculiar manner.

The *echinococci* (Fig. 52) are minute ovoid animated beings, just visible to the naked eye. When magnified they are found

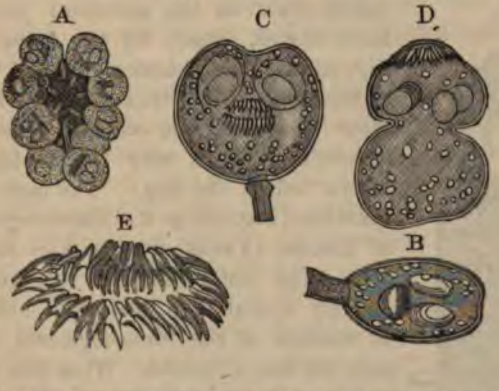

FIG. 52. Human echinococci. A. A group of echinococci, still adhering to the germinal membrane by their pedicles, magnified 40 times. B. An echinococcus magnified 107 times; the head is invaginated in the caudal vesicle; a pedicle is attached to it. C. The same compressed; the head retracted, the suckers and the hooks are seen in the interior. D. Echinococcus magnified 107 times; the head is protruded from the caudal vesicle. E. Crown of hooks magnified 350 times.—(After Davaine.)

to consist of a head resembling that of a tapeworm, provided with four suckers and a double crown of hooks (E). When the head is stretched out (D) it is seen to be connected by a short thick neck to a "caudal vesicle," which is somewhat larger than the head. The head is generally retracted within this caudal vesicle; and then the little body assumes a spheroidal figure with the crown of hooks in its interior (B C).

The echinococci are developed on, or rather in, the germinal membrane. They grow in groups of six to ten individuals,

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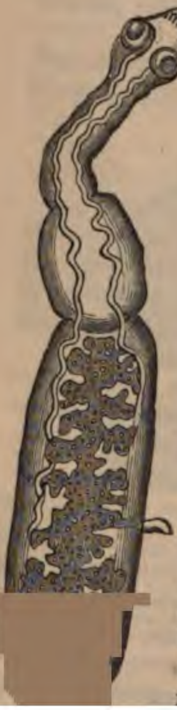
Both the echin



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where it is arrested. Having thus lodged itself, it presently reappears as a hydatid vesicle, in which, finally, are developed the echinococci as before explained. Dogs in their turn become infested with the corresponding tænia by feeding on the offal of slaughtered sheep, pigs, &c., which had been infested with hydatids. The echinococci therein contained develop in their intestines into the tænia echinococcus: and so the circle of transformation and development recommences.[*]

In the records of medicine may be found some seventy or eighty instances in which hydatids existed in the kidney or were passed by the urethra. In a number of these, the fact is simply mentioned; but in sixty-one cases some fuller details are communicated, and from an analysis of these the following account is drawn up.

It is necessary to remark that when hydatids are discharged by the urethra, it may be assumed as almost certain that they are derived from a cyst situated in the kidney. In the great majority of the cases, proof of this was obtained either from the examination of the body after death, or from the plain indication of the symptoms during life. In some cases, however, this was not so; and it remained open to conjecture whether the parent cyst was not situated in the vicinity of the ureter or bladder, and opened directly into those channels. Such an occurrence seems, however, extremely rare, and I have only been able to find one instance in which actual proof of this was obtained.[†]

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[*] For further information and details of experiments see—Gervais and Van Beneden, T. ii., p. 270 et seq.; Davaine, l. c. Synopsis, 7 and 24; and Siebold's memoir on tape and cystic worms, bound with the 2nd vol. of Küchenmeister's Manual of Parasites. Syd. Soc.'s Translation.

[†] In the Med. Times and Gaz. for 1855, i., p. 161, a case is referred to, on the authority of Mr. Birkett, in which hydatids were withdrawn by catheter from the bladder. After death a large hydatid tumour was found between the bladder and rectum, pressing upon the neck of the former. Rayer (l. c. iii. 354, foot-note) relates an instance in which a hydatid tumour in the left iliac fossa opened into the rectum, with expulsion of hydatid vesicles with the stools and discharge of pus and gas by the urethra. He cites another (p. 554, note), in which hydatids were passed by stool, and afterwards a large hydatid escaped by the urethra: but there is no information as to the seat of the cyst, the patients having recovered. There is another case, recorded by Mr. Fynney in an appendix to the 2nd vol. of the Memoirs of the Medical Society of London, in which hydatids were passed with the urine from a cyst which in all probability existed between the bladder and rectum. Immediately before the discharge of the vesicles the patient felt something give way in the neigh-

Morbid Anatomy.—The left kidney is more frequently the seat of hydatids than the right: out of 41 cases, the left kidney was affected 22 times and the right 17 times, and both organs together only twice. The less liability of the right kidney depends probably, as Béraud suggests, on the larger bulk of the liver intercepting a greater proportion of the embryos which travel from the intestine rightwards, than the smaller bulk of the spleen does of those which travel leftwards. In rare instances, hydatids have been found in the liver and other organs as well as in the kidney.

As a rule, the cyst is lodged in the substance of the kidney; sometimes, however, between the capsule and the gland. As the cyst grows it encroaches more and more on the renal tissue, and eventually may entail total destruction of the organ. It forms a roundish, elastic, fluctuating tumour, projecting from the surface of the kidney, and varying in size from an egg to an adult's head.

The cyst has a natural tendency to make its way toward the pelvis of the kidney, and discharge its contents by the ureter. When it is situated in the pyramidal portion, this event takes place early, before the cyst has attained any great dimensions: but, when situated in the cortical part, or beneath the capsule, the cyst may exist for years, and grow to a large size, before it bursts into the infundibula. It may even not burst at all; and, still more rarely, it may penetrate upwards into the chest and be evacuated through the bronchi, or open into the intestines and be discharged by stool. Sometimes, after opening in one direction, it effects a second opening in another direction. In no instance on record has the cyst burst into the peritoneum. The following table exhibits the relative frequency of these various modes of opening in our 61 cases:—

The cyst opened into the*:—

Pelvis of kidney . . .	in 46 cases	Hydatids discharged by the urethra.
Pelvis of kidney and lungs .	. 1 ,,	
Pelvis of kidney and intestines	. 3 ,,	
Pelvis of kidney and stomach	. 1 ,,	

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bourhood of the bladder. The patient died in a few weeks; but the exact seat of the cyst was not verified by *post-mortem* inspection. Cases of this class can be distinguished from renal hydatids by manual examination through the rectum or vagina.

[*] No authenticated cases exist of a hydatid cyst of the kidney opening in

Lungs alone	1 ,,	No hydatids discharged by the urethra.
Did not open at all	8 ,,	
Opened artificially	1 ,,	

Hydatid cysts of the kidney, like hydatid cysts elsewhere, are liable to certain accidents. They may contract adhesions to surrounding parts; occasion inflammation and abscess in their vicinity, and the cyst may burst into such an abscess. The cyst itself may suppurate; or it may perish, and its germinal membrane and echinococci be destroyed; the fluid it contains may be absorbed, and the whole crumple up into a hard depressed nodule, which henceforth lies dormant and obsolete. This obsolescence may ensue without bursting of the sac, or it may follow complete evacuation of its contents. The contraction and obsolescence of a hydatid cyst are accompanied by deposition of a whitish cretaceous and sebaceous material between it and the adventitious capsule, and within its own cavity. This deposit was formerly erroneously supposed to be of a tuberculous nature. Under the microscope it is found to consist of amorphous phosphate of lime, crystals of triple phosphate, cholesterine plates, and fatty granules. Amid this *débris*, echinococci hooks and shreds of laminated membrane may be found.

Hydatid cysts are also liable to external violence, especially when they form a palpable tumour in the flank. A blow or fall has in more than one instance been the apparent cause of the bursting of the sac into the pelvis of the kidney; and the patient has dated his symptoms from the occurrence of some such accident.

The opening of the cyst into the pelvis of the kidney is soon followed by the passage of secondary or daughter vesicles along the ureter into the bladder, from which they are expelled sooner or later with the urine.

The first of the two following cases illustrates the ordinary

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the loins. Rayer (iii. 578) mentions two examples of hydatid cysts in the loins which suppurated and burst externally in the lumbar region. He seems to infer that the cysts in these cases were connected with the kidney: both ended in recovery. It is more probable, however, that the cysts were lodged superficially in the muscular tissue of the lumbar region. In a later case of this kind which ended fatally, it was ascertained *post-mortem* that the cyst lay superficial to the kidney and unconnected with it.

mode of evacuation by the ureter; the second, by the ureter and lungs:—

Case I.—*Hydatid vesicles voided by the urethra, at intervals, for twenty years, with symptoms resembling nephritic colic. Hydatid cyst found in the left kidney.* (*Chopart, l. c., p.* 78.)

A young lady of 25 was seized with a violent pain in the left lumbar region, with all the symptoms of nephritic colic. There was difficulty of micturition, tension and tenderness of the abdomen. The bladder was full of urine, but some obstruction prevented its flow; though there was constant desire to pass it. In the course of the night the emptying of the bladder was effected, with discharge of a large number of hydatids. When the discharged vesicles were examined on the following morning, the majority were found ruptured, and consisted of loose membranes only; some were entire, and contained a turbid fluid. The patient was relieved by the evacuation; but the pain returned again in less severity two days after. This pain commenced in the kidney, and when it diminished in that organ it increased at different points in the course of the ureter, and became more acute at the entrance of this canal into the bladder. When the hydatids had reached the bladder the pain in all these parts was replaced by a sort of lassitude.

The patient stated that she had been subject to similar attacks for twenty years; and that they always terminated in a discharge of little bladders full of water. Some of these were as big as a pigeon's egg; others were much smaller; the latter always came away first. The attacks recurred at irregular intervals; she was sometimes six months, a year, two, and even three years without an attack. In some of the attacks the efforts at micturition would be long unavailing, until at length, by increased effort and pressure on the belly, the hydatids would shoot out with a sort of noise, and then the urine followed in full stream. Four years later, the patient died; it is not stated from what cause. The left kidney was found converted into a thick and firm hydatid sac, filled with vesicles. The pelvis and ureter were greatly dilated. The right kidney was healthy.

Case II.—*Hydatid cyst of the right kidney, which opened first into the ureter and subsequently into the right lung. Hydatid vesicles discharged with the urine and by coughing, Béraud, l. c., p.* 63.

Madame B., æt. 54, had experienced, for several months, pains in the right lumbar region and occasional difficulty of micturition; otherwise the health was good.

On Aug. 30, 1851, she was suddenly seized with such violent pain in the right kidney that she was obliged to be carried home. M. Flaux, who was called to the case, found extreme distension of the bladder; a catheter was introduced, and the urine withdrawn presented nothing unusual. The patient passed a good night, and was quite restored in a couple of days.

On Sept. 15th, she went to St. Denis, where she was seized with the same symptoms as before. She succeeded, after great efforts, in expelling by the urethra a little membranous vesicle as big as a pigeon's egg, and immediately afterwards she passed abundance of water, and was relieved.

On the 26th, the pains returned; they commenced in the right lumbar region, and radiated toward the pelvis and the right thigh. She tried to pass water several times during the night, without success. The bladder reached almost to the umbilicus. A large quantity of clear urine was withdrawn by catheter with immediate relief.

From the 8th to the 23rd of Oct., retention of urine recurred on three occasions, and the urine withdrawn did not present any peculiarities.

On Nov. 2nd, the pain in the kidney returned with great severity; it mounted to the liver and descended along the ureter to the thigh; there was thirst, hot skin, with tenderness and meteorism of the abdomen. The patient had passed water several times during the night, but in very small quantities. The urine was turbid, with a glairy deposit at the bottom of the vessel. From this date to the 22nd, the pain diminished; the urine continued turbid and contained pus.

On the 24th, the patient had a violent rigor, and the renal pain became more severe than ever. Vomiting occurred several times during the night, and three or four liquid stools were passed. She also voided urine several times. M. Fiaux now observed, for the first time, in the urine, shreds of membrane having the characters of hydatids. On examining the right flank, an oblong tumour was found below the liver, apparently united to it, extending to the iliac fossa and having a breadth of about 4½ inches. The tumour was hard, and tender on pressure; no loop of intestine passed in front of it; the lumbar region behind presented a tolerably prominent bulging. It was no longer doubtful that this was a hydatid tumour of the right kidney in a state of inflammation.

From this time hydatid *débris* continued to be discharged with the urine from time to time, and the lumbar fulness became more pronounced.

On the 22nd of Dec., under the advice of Gendrin, steps were taken to open the tumour, and several caustic issues were established on the front of it.

But on the 2nd of Jan. violent pain set in at the base of the right lung, with cough, mucous expectoration, and fever. Frequent shivering occurred the next day, and the pain and fever continued.

Jan. 7th. The oppression was increased. Violent fits of coughing occurred, with abundant purulent expectoration, of a fetid urinous odour; and this was mingled with membranes similar to those discharged with the urine. She continued to cough up hydatid shreds and urinous pus and to become gradually weaker until Jan. 22nd, when she died in a fit of suffocation, after having discharged seven or eight hydatids.

Autopsy. The small intestine was thrust to the left: the ascending colon bordered the tumour, and was intimately connected therewith in its lower two-thirds. The right lung was indurated at its base, and united to the diaphragm. Behind the cyst was a purulent collection, as large as an orange, which communicated with the cavity of the cyst. The liver, left lung, and stomach were healthy.

The tumour was found adherent to the lower surface of the liver. It was constituted by the right kidney, which was converted into a sac as large as a child's head. Few remains of the renal tissue were found. On cutting open the sac, it was found to communicate by two distinct openings with the dilated pelvis of the kidney and the abscess. The latter again, which occupied the vault of the diaphragm behind the liver, communicated by a perforation through the diaphragm with a ragged cavity in the base of the right lung. All these cavities, with the pelvis of the kidney, the bladder, and the bronchi, contained a purulent fluid and numerous hydatid vesicles.

In rare cases, the secondary cysts contain a tertiary series (grand-daughter cysts). Baillie mentions such an instance in the body of a soldier, whose kidney was found to contain a large hydatid cyst. Some of the secondary cysts in this instance merely contained fluid; others contained small vesicles floating in their interior.*

Occasionally, crystals of uric acid have been found adhering to the expelled hydatids; and in Mr. Barker's case, to be presently related, Mr. Queckett found in the interior of some of the cysts crystals of triple phosphate, uric acid, and oxalate of lime. In four cases calculi were found with the hydatids in the kidney or bladder; or were passed by the urethra.

The *symptoms* differ essentially according as the cyst has forced a passage for its contents into the pelvis of the kidney, or elsewhere, or still maintains its integrity. In the latter case the cyst remains wholly latent until it attains sufficient bulk to form a palpable tumour in the flank. As the tumour grows, it displaces the viscera in its neighbourhood, generally without further mischief; but sometimes inflammatory adhesions or suppuration take place in its vicinity and occasion intercurrent attacks of pain and feverishness. In seventeen out of our sixty-one cases, tumour in the side was discernible during life. It varied in size from an orange to an adult's head, and presented a rounded form and an elastic feel. In some instances fluctuation was distinctly perceived in it; in others obscurely; in others not at all. The peculiar thrill characteristic of hydatid tumours (hydatid fremitus) was observed only in a few instances. In order to evoke this sign, the fingers of the left hand should be laid upon the tumour, and tapped sharply with the fingers of the right. A thrill is then communicated to the overlaid fingers, which has been compared to the vibrations of a repeater watch held in the hand. A similar sensation is communicated to the ear when the stethoscope is applied and the tumour tapped with the fingers.[*] Sometimes the fremitus is absent under conditions which appear favourable to its production. In a case reported by Livois (cited by Béraud) even Rayer was unable to detect anything beyond ordinary fluctuation, and diagnosed a hydronephrosis. After death the kidney was found converted into an enormous hydatid sac containing multitudes of secondary vesicles, varying from the size of a grain of millet to a hen's egg.

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* Baillie, Morbid Anat., 5th ed, p. 294.

The topographical characters of the tumour agree with those of renal tumours in general. The colon is usually found in front of the intumescence: but it is important to know that this is not invariable. Béraud communicates a case from Nélaton's clinique, in which the descending colon ran along the outside of a hydatid tumour of the left kidney: in Fiaux's case, already related (p. 480), the ascending colon coursed along the inner border of the tumour, and no intestine separated it from the abdominal parietes.

When the cyst bursts into the pelvis of the kidney, the escape of its contents by the urethra constitutes a capital symptom. This may occur with or without symptoms referrible to the renal region (tumour, nephritic colic, &c.). Entire vesicles mixed with broken ones are usually voided; in other cases only fragments are passed, or a milky detritus in which echinococci-hooks, laminated shreds, and oil particles may be detected by the microscope.

The discharge of vesicles takes place in paroxysmal attacks, at wholly irregular intervals. In exceptional cases, only one paroxysm is experienced, during which the cyst is seemingly entirely evacuated, and then finally contracts. In the great majority of cases, however, the first attack is succeeded by many others. The interval between them may be a few weeks, or a few months, or many years. In a case reported by Tomowitz, the second attack occurred three years after the first. In Quinquerez's case, seven years elapsed between the first and second discharge of hydatids; then the attacks followed each other more frequently, at intervals of one or more years, for ten years; in the last year they recurred every four or six weeks.

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[*] The history and theory of the hydatid fremitus may be found discussed at length (with an account of Davaine's experiments) in Meissner's Beiträge zur Lehre von dem Vorkommen des Echinococcus, etc.—Schmidt's Jahrb., Bd. 116, p. 183.

An attack is usually ushered-in by sharp pain in the loin, sometimes with a sensation as of something giving way internally. The pain shoots down along the ureter to the inside of the thigh. It may be attended with rigors, sickness and hiccough —though this is rare; then follow colicky spasms in the course of the ureter, indicating the descent of vesicles along that canal —sometimes aggravated by suppression of urine and retraction of the testicle. These symptoms continue a few hours or several days, and then commonly cease suddenly, often with a feeling as if something had dropped into the bladder. The urethra is next forced, and new symptoms arise—retention of urine, excessively frequent desire to pass water, with severe pain extending to the end of the penis. When the vesicles are expelled, relief follows. The number of vesicles discharged during an attack varies from one or two to several dozens. The urine is often tinged with blood or mixed with pus. The force required to effect the final expulsion is sometimes sufficient to propel the vesicle a considerable distance with an audible thud.

The paroxysms are sometimes determined by some evident exciting cause, such as a blow or fall, or by horse or carriage exercise. In Zinkeisen's case the attacks usually followed the use of spirits and strong coffee.*

After each discharge of hydatid vesicles the tumour (if any exist) may subside sensibly. On the other hand, rapid enlargement of the tumour, from distension of the pelvis with accumulated urine, may follow the impaction of a vesicle in the ureter. Repeated discharges occasion dilatation of the passages, and enable the patient to void larger vesicles with less pain.

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* Schmidt's Jahrb., Bd. 116, p. 200.

The following examples illustrate the eccentric course and usual symptoms of renal hydatids :—

CASE III.—*Repeated discharge of hydatid vesicles with the urine : tumour in the left lumbar region—final recovery.* (*Lettsom, Memoirs of the Medical Society of London, Vol. II. p.* 32.)

A gentleman, aged 32, was thrown off his horse in Feb. 1780, and received an injury in the lumbar region. This was followed by considerable hæmaturia. In a fortnight all the consequences of his fall had disappeared; but in the following June he spit blood ; this also passed rapidly away. Three years later, he was seized with shivering and a violent pain in the left lumbar region. A few days after, he perceived an enlargement in the hypochondrium. This increased gradually until February, 1784. After the first month the tumour was so little painful that he was enabled to take a journey of 130 miles to London to consult Dr. Lettsom.

A fluctuating tumour as large as an infant's head was detected in the left hypochondrium, extending from the spine to the umbilicus and from the ribs to the os innominatum.

As the swelling augmented the pain increased, and the patient suffered considerably from the action of walking and from motion in general. At length (Feb. 20th) some difficulty in making water was experienced, and for many hours there was a total obstruction of urine. The same night there was great pain with violent rigors ; but early in the morning the patient experienced the most happy relief by the discharge of a large quantity of thick pus with the urine, which was followed the next day by the escape of numerous hydatids.

In a few days the tumour subsided, and the purulent discharge ceased; after this, he continued recruiting in health for nearly a fortnight, when his side enlarged again, after exercise in a coach, probably by a large hydatid stopping up the ureter ; rigors and strangury succeeded as before, and the tumour became as large as in the first instance, until the latter end of March, when he experienced a second discharge in every respect like the former, excepting that the hydatids were much larger.

His health and strength again returned, until his side filled a third time, after exercise on horseback, and continued swelling until the 25th of April, when he was again relieved by a third discharge ; the hydatids now passed were considerably larger than those of the preceding attacks.

The passages now became so open, that he frequently discharged hydatids after walking or riding, without enlargement or pain of the side ; or if he felt uneasy or perceived a tendency to tumescence, by pressing his hand upon the side, he could squeeze the vesicles into the bladder, where they would remain some time before they were discharged ; but the hydatids became at length so considerable in size that it was with great difficulty they passed the urethra. The last vesicle which he voided (on the 12th of July) was so very large that it stopped up the urethra, and remained in it for a considerable time, until the weight of the accumulated urine forced it away.

The earliest hydatids voided burst in their exit ; and they gradually increased in magnitude in every successive discharge; the first which he passed were not bigger than the skin of a green pea, and the last about the size of a pullet's egg.

Since this last discharge his health was gradually re-established; he was able to enjoy, without the least inconvenience, thereafter, the chase and every other species of exercise as well as ever he did.

CASE IV.—*Discharge of hydatids by the urethra in periodical paroxysms, occurring yearly for a period of thirty-seven years.* (*Vigla, Bulletin de la Société Anatomique*, 1838. *Cited by Béraud, l. c. p.* 57.)

A healthy woman, æt. 37, had suffered from her infancy with her present symptoms, which occur in annual paroxysms. Every winter, and generally in the month of January, she experiences in the left renal region a pain, which speedily becomes severe and forces her to relinquish her occupation. There is no fever; nor vomiting; but the appetite is lost; the urine remains natural. At the end of two or three days of this condition, she voids a very large number of hydatids, mingled with a turbid urine. This emission takes place two or three times a day, for three or four days, and then she returns to her ordinary health. Sometimes, but very rarely, similar attacks occur in the course of the year, but slighter; these latter consist of a violent pain in the same place, not lasting more than two or three hours, at the end of which an emission of urine with discharge of hydatids ensues, and the pains disappear. These slighter attacks have never recurred more than once or twice in the same year. In the year 1838 one of the annual January attacks was observed by M. Vigla. The prodromata, that is to say the pain and uneasiness, were accompanied with feverishness; but this was attributed to a co-existing acute pulmonary catarrh. At the end of four days, as usual, the emission of urine charged with hydatids commenced, and continued for four days. The quantity of vesicles which she rendered was enormous; for she passed urine twice or thrice on each of these three days, and every time from 40 to 50 large hydatids were found in the urine, without counting the little ones. The larger ones passed the first, but ruptured and empty; the largest of all surpassed the size of a pigeon's egg. The smaller ones were voided entire, and full of a semi-transparent fluid; some were smaller than a pea. There were no symptoms referrible to the bladder.

CASE V.—*Hydatid cyst of the right kidney; suspicion of pregnancy; discharge of hydatid vesicles by the urethra.* (*Babington, Med. Times and Gaz.*, 1855, *I. p.* 160.)

A healthy single woman, æt. 27, was admitted into Guy's Hospital on Feb. 8th, 1854. About the age of 23 she was one day kicked in the abdomen by a child which she was carrying up-stairs. The kick gave her much pain, and on the night following she discovered for the first time a tumour about the size of an egg in her right side. The tumour gradually increased, and in the course of a year became so large as visibly to distend the abdomen. About this time the menstrual function was suspended, and the increasing size of the abdomen caused her great trouble, by exciting suspicions in the minds of her relatives that she was pregnant. The enlargement, however, continued beyond the usual period of utero-gestation, and anxieties as to the nature of the disease took the place of the suspicions alluded to. She was now sent up to London from her home in Oxfordshire, and was admitted into St. Bartholomew's Hospital, under Dr. Hue. The tumour was at this time stationary, and her general health good. After a few weeks' stay in the hospital she was discharged, and returned to service, where she continued without material change in her condition until about a year prior to her admission into Guy's Hospital, under Dr. Babington, when she began to pass "skins and little bladders," with the urine. These bodies continued to be voided afterwards in large numbers. Often a vesicle would get impacted in the urethra, and require to be pulled out with the fingers. At first neither blood nor matter was ever present in the urine. About two weeks before admission, however, after having been confined to bed for several days with intense pain in the side, she suddenly felt a sensation as if something burst within her, and shortly afterwards matter and blood began to escape by the urethra. The tumour had meanwhile much diminished in size, and at the time of her admission there was no visible enlargement of the abdomen. During her illness she had lost some flesh, but still retained a fairly robust appearance.

On examination of the abdomen, a large mass apparently about the size of a fœtal head, but flattened, was easily felt in the right hypochondriac or lumbar region. It was not tender, and felt firm. The patient remained under Dr. Babington's care for several months, during which vast numbers of hydatid vesicles were passed. The vesicles varied much in size, some were broken and others whole. The urine contained also much pus. The girl somewhat improved in health, and the tumour became decidedly smaller before she left the hospital; at the time of her discharge, she still continued to void occasionally pus and hydatid vesicles.

CASE VI.—*Frequent discharge of hydatid vesicles by the urethra—nephritic colic and suppression of urine. No tumour in the flank.* (*Dr. Barker, l. c. p.* 5.)

A young man, æt. 28, came under the notice of Dr. Barker, of Bedford, on Dec. 17th, 1853. He was suffering from a dull aching pain in the loins, particularly on the left side, with frequent desire to pass urine, and slight difficulty in voiding it. The urine was healthy. On the 22nd of Dec. he experienced greater difficulty than ever in passing urine in the early part of the night, and for some hours he was unable to pass a single drop. Early in the morning he passed four little hydatid cysts with immediate relief. Subsequently he recovered sufficiently to follow his occupation during the summer of 1854, suffering nothing more than an occasional frequent desire to void urine.

On Sept. 10th, 1854, he passed six cysts; but with less pain than on the previous occasion—a result which the patient attributed to taking 10 drops of oil of turpentine, which had been recommended to him, and which greatly increased the diuresis. The urine after the passage of the cysts was tinged with blood.

On Nov. 16th he passed four cysts. The passage of these was preceded by severe pain in the left kidney, by the passage of several pieces of clotted blood, and by considerable difficulty in voiding urine. Indeed, for two entire days he passed no urine. On this occasion he took 19 drops of turpentine, within two hours, in divided doses. Shortly after taking the turpentine the pain in the left kidney suddenly ceased, with a sensation which, to use the patient's own words, seemed to indicate that "something had suddenly broken in the kidney." He then complained of pain along the left iliac region, which continued for several hours, and ceased as suddenly as the previous pain had done. After this, all attempts to void urine were accompanied with pain along the urethra, premonitory to the expulsion of the cysts from that passage.

He continued in good health, with the exception of occasional dull aching pain in the lumbar region, especially the left side, until Dec. 9th. He then passed five cysts, but all smaller than the previous ones; and no more were passed until Dec. 31, when he awoke in the morning with acute pain in the loins, and all the symptoms previously described as occurring on Nov. 16th. During the day he passed twenty cysts—one at 8 a.m.; eleven at 1 p.m.; five at 7 p.m.; and three at 11 p.m. The cysts passed in rapid succession, and some were of a size as large as a small walnut. On Jan. 1, 1855, a single cyst was passed in the morning; on the 2nd two others; on the 3rd one, and on the 10th two. From this last date up to Dec. 8th (beyond which the history is not carried), he continued to suffer frequently from attacks of pain and difficulty in passing urine, followed often by the expulsion of cysts, between seventy and eighty of which he brought to Dr. Barker.

Careful examination failed to detect any abdominal enlargement.

The urine often contained a small quantity of blood during and after the expulsion of the cysts; it was often loaded with lithates and phosphates; occasionally, crystals of uric acid were found attached to the outer surface of the cysts. The general health suffered little. Altogether upwards of 150 cysts were passed; they varied in size from a pin's head to a walnut. The larger vesicles contained echinococci; but many of the smaller ones did not contain any.

The *duration* of the symptoms is altogether uncertain. In some of the cases permanent recovery followed one or a few discharges of vesicles. Other patients went on passing hydatids for three, ten, twenty, and even thirty years. A discharge of vesicles having once taken place, there are no means of ascertaining whether any more attacks will follow. Neither the number of vesicles voided, nor the frequency of the discharges supply any reliable indication. The only sign of value is the lapse of time since the preceding attack: the longer the interval the less probability of recurrence.

The usual *termination* is recovery. Out of sixty-one cases, recovery was assumed to have taken place in twenty; in most of these the attacks had ceased for some years. In sixteen cases, vesicles continued to be discharged at the date of the record; in nineteen cases the termination was fatal; and in six we are left without information. Of the nineteen fatal cases, death took place in nine from causes other than the hydatid disease (phthisis, cancer, gangræna senilis, &c.); so that only in ten (sixteen per cent.) was the fatal issue attributable to the parasite. Death was brought about in these ten cases in diverse ways—by bursting of the cyst into the bronchi, by pleurisy from pressure of the tumour on the thoracic cavity, suppuration of the sac, &c. In a case reported by Dr. Blackburn, the left kidney was the seat of a hydatid cyst which had burst into the pelvis of the organ, where a large calculus was also found; the right kidney was congenitally absent; so that the abrogation of the function of the left (and unique) kidney proved necessarily fatal.*

Etiology. Hydatids are not uncommon in England, France, and Germany: more rare in America and India. There is, however, no country so fearfully infested therewith as Iceland. According to Eschricht (speaking of hydatids in all parts of the body), a sixth part of the population are afflicted with this parasite. The frequency of the disease is due to the vast number of dogs in that country, which live in intimate contact with the inhabitants, and are greatly infested with the tænia echinococcus. The ova of the parasite, discharged with the excrements of the dogs, foul the dried fish which forms a large part of the food of the population. The embryo of the parasite thus finds its way into the stomach, and thence travels into different parts of the body, giving rise to hydatid cysts.

The use of uncooked meat and salad is evidently an easy source of infection, in places where dogs are numerous and live in close intercourse with their masters. Dr. Barker's patient had been for a year a vegetarian.

Men appear more subject to renal hydatids than women—the proportion, in our sixty-one cases, was forty men to twenty-one women. In only one instance were more than one member of a family affected: in that case, a husband and wife passed hydatids by the urethra.†

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* Lond. Med. Journ., 1781, p. 126.

† Gay, Med. Times and Gaz., 1855, I. 160.

The mean age, in forty-five cases, was thirty-four years: the youngest was only four years, and the oldest seventy-five.

The *diagnosis* presents no difficulty when a tumour exists in the side and hydatid vesicles are voided with the urine. When the vesicles are broken in the passage, the laminated structure of the pieces, or the finding of echinococci-hooks, decides the nature of the discharge.

So long as the parent cyst remains intact, the urine preserves its normal characters, and the diagnosis turns on the characters of the tumour in the flank. Hydatid fremitus, when present, (which is rare) is a valuable sign; but its absence, as we have seen, has little significance.

Hydatid tumour of the kidney is most liable to be confounded with hydronephrosis; and in the absence of discharge of vesicles, or their *débris*, with the urine, and of hydatid fremitus, the diagnosis is extremely difficult, or impossible: it rests chiefly on the indications of the previous history.

When vesicles are voided with the urine, and no tumour can be detected in the flank, the seat of the parent cyst is sometimes indicated, quite clearly, to be the kidney, by signs of nephritic colic—in other cases, more obscurely, by pains in the back and loins or about the crest of the ilium. When these indications fail, a careful examination of the pelvis should be made through the rectum or vagina: if no evidence be found of a tumour between these parts and the bladder, it may be inferred, almost with certainty, that the parent cyst is situated in the kidney.

The *prognosis* is generally favourable—much more so than in hydatid cysts of other internal organs (the uterus excepted), on account of the facility and safety of evacuation by the urinary passages. It is most favourable of all when the discharge of hydatids by the urethra is unassociated with tumour in the abdomen. In no such case has a fatal result been recorded: the cyst in such cases may be inferred to be small, and to be situated in the pyramidal structure of the kidney, whence its contents find easy exit through the infundibula.

When a renal tumour exists, the issue is still likely to prove favourable if the cyst has opened into the urinary passages. There is, however, some risk that a second opening may be formed in a less safe direction (into the lungs), or that the cyst, or the parts around, may suppurate. This latter contingency is by no means rare, nor is it necessarily fatal. In several instances large quantities of pus were discharged with the vesicles, and yet the issue was favourable. In three cases, in which vesicles were discharged both by stool and with the urine, the termination was favourable. In an instance recorded by Fleckles, a woman who had had a tumour in the side for many years voided frequently hydatids by the urethra, and subsequently a large quantity by vomiting. At the date of the report the case was going on favourably.* In the two cases in which the cyst burst into the cavity of the thorax, the termination was fatal.

When the cyst fails to open a passage for its contents into the pelvis of the kidney, the prospects of the patient are much more serious. The tumour is liable to attain very great dimensions, and, by its pressure, to excite inflammation in the surrounding parts, or within the chest; or the cyst itself may suppurate and be transformed into a vast abscess. The operation of puncturing such a cyst is one of considerable danger.

Treatment. The indications to be held in view are, to destroy the life of the parasite, to facilitate the evacuation of the cyst, and to combat the accessory symptoms and complications.

Whether medicines administered internally have any real power to destroy the life of a hydatid parasite, or to facilitate the evacuation of a hydatid cyst, may be greatly doubted. Nevertheless, oil of turpentine has obtained a certain reputation on the strength of its tænia-fuge properties. The echinococcus of the hydatid vesicle is undoubtedly identical with the head of a certain species of tape-worm; but the condition of a parasite free in the intestinal canal, is widely different from the encysted state of the same parasite in the substance of the kidney, where remedies can only reach it indirectly, by the circuitous route of the circulation. Turpentine was given in a large proportion of the recorded cases; but there is little evidence that it had any beneficial influence beyond its diuretic effects.

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* Schmidt's Jahrb., Bd. 87, p. 205.

The escape of the vesicles in Dr. Babington's case, was thought to be favoured by a course of iodide of potassium.

A variety of other vermifuge and diuretic medicines have been used, with more or less show of success—calomel, nitrate of potash, the caustic alkalies, hemlock, taraxacum, etc. Béraud states of his patient, that whenever he took white wine, and beverages containing nitre, he voided a much larger number of vesicles than at any other times. On two different occasions he was made to take 20 grains of nitre in dandelion tea, and each time the desired effect was speedily produced.*

Electro-puncture has also been practised with a view to kill the worm; but without evidence of success.

When the cyst has opened into the pelvis of the kidney, the practitioner is able, in diverse ways, to facilitate the expulsion of the vesicles, and to moderate the severity of the accompanying symptoms. Anodynes, especially opium, the warm bath, free use of diluents, are indicated during the passage of the vesicles: if the nephritic paroxysm be intense, blood may be abstracted from the loins by cupping. Sometimes mechanical aid is required to assist the liberation of the vesicles. Dr. Lettsom's patient (see p. 485) helped their transit along the ureter by pressing them forward with his fingers; and in several cases it is noted, that patients (mostly women) have used the fingers to dislodge vesicles impacted in the orifice of the urethra. The use of the catheter is sometimes required, to relieve the retention of urine caused by vesicles engaged in the urethra or pressing against the neck of the bladder.

When the cyst remains closed, measures may be taken (as in similar cysts of the liver) to evacuate its contents by artificial puncture. In renal hydatids, however, this proceeding, necessarily one of considerable danger, should be delayed as long as possible, in the hope that spontaneous rupture may yet take place into the pelvis of the kidney, and the contents be more safely evacuated through the urinary channels. Operative procedures are only justifiable when the cyst has attained great dimensions, and is exercising dangerous pressure on the thoracic organs, and threatening to burst in some untoward direction.

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* Gaz. d. Hôp., Aug. 11, 1832. Béraud, l. c., p. 93.

If operation be decided on, it should always be preceded by an exploratory puncture with a fine trocar. If the tumour bulge in the loin, it should unquestionably be opened from behind, in order to avoid the peritoneum. If it bulge forward, the safer plan is probably to proceed according to the method of Recamier, and procure previous adhesion of the peritoneal surfaces to the sac, by means of a succession of caustic potash applications, before penetrating into the cavity. When this is accomplished, the cyst may be evacuated by a larger trocar, and afterwards injected with iodine solutions.

In a hydatid cyst (probably of the liver) successfully treated by Mr. Cock, repeated punctures with a needle trocar were made at intervals of about ten days. From two to four pints of limpid fluid were removed on each occasion. After the third puncture, the evacuated fluid became turbid and brownish. After the fifth puncture, the fluid was withdrawn about every third day, and the cavity washed out with warm water for a period of about three weeks. A fortnight later, the opening was enlarged by bistoury, and a large quantity of hydatids evacuated. The cyst gradually contracted, and suppurated; it was occasionally washed out with a solution of chlorinated soda when the discharge became offensive. Complete recovery took place in ten months. (Med. Times and Gaz., 1855. I. p. 57.)

II. BILHARZIA HÆMATOBIA—*Cobbold.*

(*Distoma Hæmatobium—Bilharz.*)

BILHARZ—Zeitschr. für Wissenschaftliche Zoologie. Bd. iv.
GRIESINGER—Beobachtungen über die Krankheiten von Egypten. Archiv. d. Physiolog. Heilk. 1854, p. 561.
DAVAINE—Entozoaires. Synopsis, No. 38.
COBBOLD—Entozoa, p. 197.
HARLEY (DR. JOHN)—Endemic hæmaturia of the Cape of Good Hope. Med. Chir. Trans., vol. xlvii. p. 55.

THIS parasite was discovered by Bilharz, while conducting, with Griesinger, an investigation into the diseases of the Egyptians. Bilharz named it Distoma Hæmatobium; but later writers have erected it into a separate genus, which

Cobbold has named Bilharzia in honour of its discoverer. It is an elongated, soft-skinned, bisexual entozoon, three or four lines in length, of the trematode or fluke kind (Fig. 54). It inhabits the branches of the portal system, and the minute veins of the pelvis of the kidney, ureter, and bladder. So common is it among the Egyptians, that Griesinger found it 117 times in 363 autopsies.

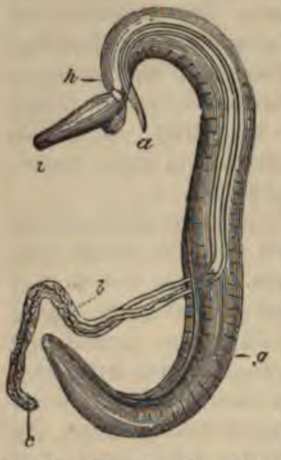

Fig. 54. Bilharzia Hæmatobia, highly magnified. *h i g*, the male; *a b c*, the female. (After Bilharz.)

The male (*h i g*) is comparatively thick and short, and provided with a gynæcophoric canal, in which the longer, filiform female (*a b c*) is lodged during the copulatory act.

The eggs (Fig. 55*b*) are oval bodies, $\frac{1}{170}$ of an inch long, with a spiny projection from the anterior end. The embryo, when newly escaped, is flask-shaped, and provided with cilia.

This creature does not produce much mischief in the larger veins; but when lodged in the smaller vessels of the mucous and submucous tissue of the urinary and intestinal tracts, it engenders severe and often fatal disorganization. Griesinger found that, in the large intestines, it gave rise to a disease resembling dysentery, and that it was a frequent complication of that disease, but not the essential cause of it.

The ravages of the Bilharzia produce much more serious results in the urinary channels than in the intestines. It chiefly affects the bladder: but frequently also the ureter and pelvis of the kidney.

In the bladder, it gives rise to injected and ecchymotic raised patches, varying from the size of a lentil to that of a shilling covered with a tough mucus, or with greyish-yellow, bloody exudation, which contains masses of ova. In more advanced stages the patches are more elevated, discoloured, mixed with pigment specks, smooth and leathery, or soft, friable, and encrusted with gravelly matter, composed of uric acid and other urinary deposits, mixed with ova and blood. In other cases, the patches resemble nodules or condylomata, over which the mucous membrane is sometimes preserved uninjured, sometimes thickened, injected, adherent, or detached.

When the parasite invades the ureter and pelvis of the kidney, its effects are still more destructive. The calibre of the ureter is narrowed at the affected spot. Above the con striction, the ureter is dilated from accumulation of urine; the pelvis is also distended, and a hydronephrotic condition is produced. Or, inflammation and suppuration are set up, and severe pyelitis ensues. In one instance, Griesinger found the kidney distended into an enormous sac filled with pus—the renal tissue being wholly destroyed.

In addition to these direct results, urinary concretions are often formed on masses of ova, and grow into large calculi. This accounts for the frequency, and endemic prevalence, of calculous disorders in Egypt.

Griesinger remarks:—"These various changes in the mechanical state and nutrition of the uro-poietic apparatus fail not to react most deleteriously on the entire organism. A series of cases have fallen under our notice, in which they produced general ill health, and, at length, death. Most of these individuals were finally cut off, with shattered constitutions, by pneumonia, dysentery, and the like. . . . . The direct signs of the disease are to be sought in the uro-poietic system, but especially in the urine. Repeated hæmaturia in sickly individuals, from unknown causes, often came before us in Egypt. We no longer doubt that the symptoms were produced by distoma-processes. The eggs of the distoma were found by Bilharz in the urine of a boy who, during convalescence from typhus, suffered from hæmaturia.* Symptoms of pyelitis, or slight affection of the bladder, must be present in many cases." . . . . "Cases also came before us, which awoke a strong suspicion, that the Distoma disease sometimes ran its course as an acute, severe, and fatal disorder. We found on two occasions, in the bodies of persons who had rapidly died from an unknown acute disease, abundant recent distoma-changes in the bladder, recent pyelitis, and a uniform dark-red hyperæmia of the kidneys. In other cases of supposed rapid typhus, the same changes were found in the bladder and ureter." These researches open a wide field for conjecture. Not only may urinary derangements and uræmia be occasioned by the ravages of the parasite, but septic infection may arise from the accumulation of heaps of dead and dying animals in the portal vessels; or the animals may creep into the general circulation, and find their way into organs of vital importance: in one instance distoma egg-shells were found in the blood of the left ventricle.

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* Griesinger states that the clinical aspect of the subject only began to engage their attention when he and Bilharz were about to quit Egypt.

In a note to his remarkable paper, Griesinger throws out the conjecture that the endemic hæmaturia of hot countries may be due to the presence of this worm in the urinary passages. A most interesting confirmation of this conjecture has been recently supplied by the researches of Dr. John Harley. Dr. Harley had an opportunity of examining the urine of three gentlemen who had resided at the Cape of Good Hope, and who had been subject to the endemic hæmaturia of that country. One of these still continued to be affected with slight hæmaturia; the other two considered themselves cured of the hæmaturia, but were subject to gravel. In the deposit from the urine of all three, Dr. Harley detected numerous ova of the Bilharzia. The condition of the urine in the first case is thus described by Dr. Harley:—"Pale-amber coloured, sp. gr. 1017·6, acid, deposits a deep layer of dirtyish-white flocculent matter, amongst which were two short opaque filaments about the $\frac{1}{35}$ of an inch in diameter, of a brownish colour and soft consistence, two shorter and wider fragments of the same substance, a little reddish mass of the size of a hemp-seed, like a little clot of blood, and numerous white specks. The clear limpid urine, when acidulated with nitric acid and heated, deposited a trace of albumen." Uric acid, oxalate of lime and urates were also sometimes found. The deposit, examined microscopically, was found to contain pus corpuscles; and the filamentous bodies and coagula contained imbedded in them great numbers—sometimes thirty or forty, or more—of bright highly refractive oval bodies, which were identified as the ova of Bilharzia (Fig. 55). These observations seem to establish the parasitic origin of the endemic hæmaturia of Cape Colony, and render it exceedingly probable, that the endemic hæmaturia of the Mauritius and other hot climates has a similar origin.

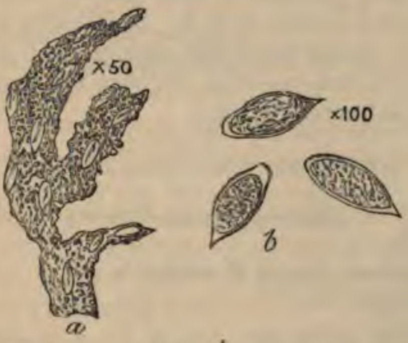

FIG. 55. Ova of Bilharzia hæmatobia, found in the urine of a patient suffering from the endemic hæmaturia of the Cape of Good Hope. *a*, filament of mucus containing ova imbedded in it × 50; *b*, ova as they appeared in the fresh urine × 100. (After Harley.)

Of the *treatment* of this parasite Dr. Harley observes :—"I have found that a draught composed of ♏xv. each of oil of turpentine and male fern, and ♏v. of chloroform, in ℥ii. of tragacanth mixture, given every morning, brought away great numbers of the ova. The saline (? acid) condition of the urine is much diminished, and the renal irritation and pain due to the presence of crystalline concretions are much relieved by the administration of bicarbonate of potash in copious draughts of water. The alkali dissolves the uric acid, which I believe to be the cementing medium of the oxalic deposits, and thus the disintegration of the calculi is facilitated, and their formation prevented" (l. c. p. 68).

From the researches of Siebold on the trematode worms, it may be assumed, that between the ciliated embryo above mentioned and the adult sexual worm, there are two other distinct forms, which serve to complete the chain of metamorphoses connecting these two extremes of development. Fresh-water mollusca and fish are probably the victims selected by the parasite during its development through these intermediate stages. Harley on these grounds suggests the following prophylactic measures in districts affected with endemic hæmaturia: 1. The water should be conveyed from its source to its destination in covered channels, so that the ova contained in the urinary and fæcal products of those infested with the parasite may be prevented mixing with it. 2. Drinking-water should be filtered. 3. Salads which may entangle small mollusca containing parasites, and uncooked molluscs and fish (as smoked fish), should be carefully avoided.

III.—STRONGYLUS GIGAS—*Rudolphi.*

(*Eustrongylus gigas—Diesing.*)

DAVAINE—Entozoaires. Synopsis 99, and p. 267.
COBBOLD—Entozoa, p. 358.

This is the largest of the nematoid worms, and in its general conformation resembles a gigantic lumbricus. The male measures from ten inches to a foot in length, and a quarter of an inch in breadth, while the female has sometimes a length of more than a yard. It is distinguished from the common round worm by its reddish colour (which is, however, apparently due to the sanguineous fluid in which it is usually bathed), its greater size, and the existence of six nodules or papillæ round the mouth. The Ascaris lumbricoides has only three oral papillæ.

This worm is almost peculiar to the kidney and urinary passages, and is very rarely found elsewhere. It inhabits weasels, the North American mink, and has been found in the dog, wolf, horse, ox, and some other animals. It is of extreme rarity in the human subject. Of the seventeen alleged cases collected by Davaine, he only classes seven as even probable instances. There are none of recent occurrence; and it is evident that most of the alleged cases were really examples of lumbrici, which had penetrated into the urinary passages from the intestines.

A very fine specimen is preserved in the museum of the London College of Surgeons, which I have had an opportunity of examining. It is an undoubted strongylus, more than a foot long. It originally belonged to Brookes's museum, and is entered in Brookes's catalogue as "an uncommonly fine specimen of an enormous worm (strongylus gigas) found in the kidney of a patient of the late Thomas Sheldon, Esq."

IV.—PENTASTOMA DENTICULATUM—*Rudolphi.*

DAVAINE—Entozoaires, pp. lxxxviii. and 293.
WAGNER—Archiv der Physiologische Heilkunde. 1862, p. 581.
COBBOLD—Entozoa, p. 394.

This is a very minute encysted parasite, about a line and a half long, club-shaped, with a double pair of hooks, and devoid of sexual organs (see Fig. 56). It is conjectured by Davaine to be the larva of pentastoma tænioides, which infests the frontal sinuses of dogs and horses. No symptoms are known to be produced by it.

The only known instance in which the parasite was found in the urinary organs is the following:—In making the autopsy of a painter, sixty-two years of age, who died of Bright's disease, Wagner found on the convex border of the right kidney a small, whitish, slightly-raised oval patch of fibrous appearance, about one-seventh of an inch long. It was situated under the capsule of the kidney. This little body was hollow in the interior: it contained a yellowish mass, which on examination disclosed the presence of a worm, which was recognised as the pentastoma denticulatum of Rudolphi.

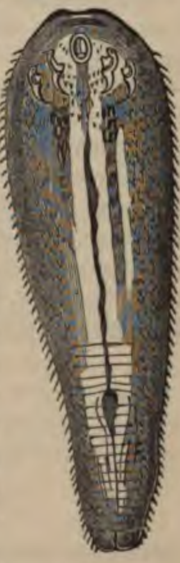

FIG. 56
Pentastoma denticulatum, greatly magnified. (After Zenker.)

This worm is common on the surface of the liver in goats, oxen, rabbits, cats, and some other animals. It has also recently been found on the surface of the liver in man, by Zenker in Dresden, Heschl in Vienna, and by Virchow, Wagner, and Frerichs in other parts of Germany. Cobbold states that Dr. Murchison, during the time he held the office of Pathologist at the Middlesex Hospital, diligently searched for it without success.

V.—ERRATIC WORMS.

Intestinal worms sometimes penetrate into the urinary passages, and are voided with the urine. In women, thread worms occasionally creep into the bladder through the urethra; and in both sexes lumbrici, and joints of tapeworm, have been known to creep into the bladder through fistulous communications, caused by abscesses, passage of pins, lithotomy, &c.

VI.—SPURIOUS WORMS.

The *spiroptera hominis* of Rudolphi, the *diplosoma crenata* of Farre, and the *dactylius aculeatus* of Curling, have been clearly proved by Schneider and Cobbold to be examples of imposition —witting or unwitting—on the part of patients. The history of the so-called diplosoma crenata of Farre furnishes one of the most remarkable examples ever put on record of long-continued and successful deception practised on scientific inquirers. The following references may be consulted on the subject:—W. Lawrence, *Med.-Chir. Trans.* vol. ii. 385; A. Farre, *Beale's Archives of Medicine*, vol. i. p. 290; A. Schneider, *Reichert and Dubois's Archiv*, 1862, p. 275; Cobbold, *Entozoa*, pp. 403, 409; Curling, *Med.-Chir. Trans.* vol. xxii. p. 274.

CHAPTER XIV.

ANOMALIES OF POSITION, FORM, AND NUMBER OF THE KIDNEYS.

CHOPART—Malad. d. Voies Urinaires. (Edit. by Ségalas.) Paris, 1855, p. 53.
RAYER—Malad. d. Reins. T. iii. 769.
DURHAM—Guy's Hosp. Reports. 1860, p. 404.
ROSENSTEIN—Nierenkrankheiten. p. 472.
VOGEL—Krankh. d. Harnbereitenden Organe. Erl. 1865, p. 706.

THE kidneys are subject, like other organs, to certain deviations from their natural situation, form, and number. Most of these deviations are congenital; others are acquired later in life, through accident or disease. Some of them are appreciable during life, and are liable to be confounded with wholly different pathological states: others are entirely latent; and are, so long as the healthy state is maintained, nowise detrimental to the subject of them; but bring greatly increased risks, under certain contingencies of obstruction to the course of the urine.

I. ANOMALIES OF POSITION.

The kidneys may occupy an unnatural situation, and remain permanently *fixed* in that situation; or the misplaced organs may possess a certain *mobility*.

A. *Fixed Malpositions of the Kidneys.*

The kidney may be displaced downwards, upwards, or laterally, by the pressure of a tumour growing in its vicinity, or by an enlarged liver, spleen, pancreas, or supra-renal body: in these cases the malposition is *acquired*. But the malposition may also be *congenital*. Instead of lying beside the vertebral column, deep in the lumbar region, the organ may be fixed in front of the vertebræ, or on the brim of the pelvis, or within that cavity ; in a case figured by Ruysch, the kidney lay cross-wise, with its hilus turned upwards, the ureter descending behind it.

A kidney congenitally misplaced, usually deviates more or less from its natural configuration, and is associated with malposition of some portion of the large intestine and peritoneum. The renal artery and ureter also, necessarily, deviate less or more from their natural distribution. The corresponding supra-renal capsule does not (in congenital cases) follow the kidney into its abnormal situation, but invariably occupies its usual place in the lumbar region.

By far the most common, and also the most practically important, of the fixed misplacements of the kidney, are those in which the organ lies within, or upon the brim of, the pelvis. In these cases the misplaced organ is liable to be felt during life, either through the abdominal wall, or the vagina, and to be mistaken for some other object ; if it lie within the pelvis, it may embarrass and complicate parturition.

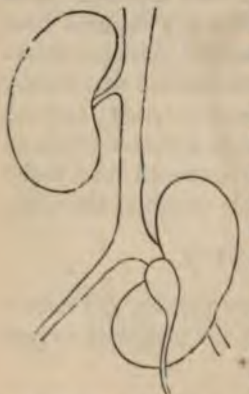

FIG. 57. Left kidney, lying on the left sacro-iliac synchrondroses.—(From a drawing in the possession of Dr. Renaud.)

In nineteen cases of congenital malposition of the kidney, which I have been able to collect and compare, the abnormality was, in every instance, confined to one kidney ; and the left kidney was much more commonly affected than the right (left 14, right 5).

The most frequent of these deviations was to find the kidney lying obliquely on the sacro-iliac synchondrosis, as represented in Fig. 57. In some of the cases, the organ was fixed beside the uterus, or transversely between the rectum and bladder, or across the prominence of the sacrum.

Mr. Canton has described and figured a curious specimen, taken from a man who died (of bronchitis) at the age of twenty-seven. There were no renal symptoms during life. The right kidney was in all respects normal; but the left was situated below, and between, the bifurcation of the aorta, as shown in Fig. 58. Instead of presenting the ordinary kidney-shape, the gland was rudely oval, and, on some parts of its surface, lobulated. The pelvis of the organ was directed almost immediately forward, and the upper portion of the ureter was dilated, owing to the impaction in it of an oxalate of lime calculus, weighing 2½ drachms. The left renal arteries were two in number, and sprang from the fore part of the aorta at a short distance above its division. The sigmoid flexure of

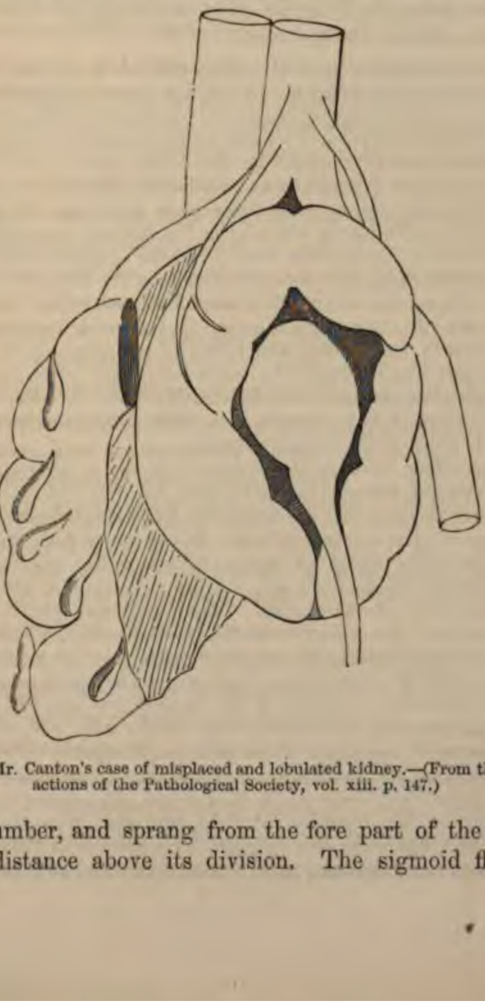

FIG. 58. Mr. Canton's case of misplaced and lobulated kidney.—(From the Transactions of the Pathological Society, vol. xiii. p. 147.)

the colon was placed, as represented in the engraving, on the right side of the kidney.

It rarely happens, that malpositions of this class produce any evidence of their existence during life; but sometimes, as in the two following cases, the misplaced organ forms a palpable tumour in the abdomen, which is liable to be mistaken for something of a more serious character; or, in the female, it may constitute an obstacle to parturition.

CASE I.—*Left kidney malformed, and situated over the left sacro-iliac synchondrosis, mistaken for an abdominal tumour.* (*Durham, Guy's Hosp. Reports*, 1860, *p.* 407.)

Mr. W. S., previously in good health, suffered, at the age of forty-five, from a severe attack of fever. During his recovery he noticed, for the first time, a tumour deeply seated in the hypogastric region, somewhat on the left of the middle line. This tumour was found on examination to be oval in form, somewhat elastic to the touch, and fixed. It was not nodulated, nor did it present any distinctive elevations or depressions. Manipulation gave rise to very disagreeable sensations, but not to acute pain. Considerable alarm was felt by the patient, especially as some members of his family had died from "tumour in the abdomen." In the course of a short time, when convalescence from the fever was established, a second opinion was taken. The conclusion arrived at was, that there existed in the lower part of the abdomen "a tumour of doubtful character." Iodine ointment was applied, and iodide of potassium taken internally. The treatment was continued for some time, but, of course, did not produce the slightest effect on the tumour. Mr. S. never thoroughly recovered his health and strength, and, about four or five years after his attack of fever, died of pulmonary disease.

Autopsy.—Upon opening the abdomen, it was at once seen that the supposed tumour was nothing more than the left kidney, which was situated over the sacro-iliac synchondrosis, and extended somewhat on the promontory of the sacrum, and also, by its lower part, into the true pelvis. The colon formed no sigmoid flexure in the left iliac fossa, but passed across the middle line; and the commencement of the rectum was on the right side of the sacrum. The supra-renal capsule was in its normal position. The kidney presented two depressions, which divided its surface somewhat indistinctly into three portions. The principal arterial supply was derived directly from the aorta, by a branch coming off just above the bifurcation; a branch of the common iliac artery of the *opposite* side, and a branch of the internal iliac of the same side, also supplied different parts of the organ. There was one principal vein, which passed from the internal and posterior part of the kidney into the vena cava just above the junction of the common iliac veins. The ureter resulted from the junction of four branches; of these, two came from the upper and posterior part, while the two principal ones came from the anterior and lower part; these branches joined one another about an inch from their several points of exit from the organ. Thus this kidney presented no distinct hilus, nor, consequently, did it possess the characteristic kidney shape. The right kidney was in its natural position, and both glands were quite healthy.

Case II.—*A misplaced left kidney offering an obstacle to parturition.* (*Hohl, cited by Rayer*, l. c., tom. iii. p. 774.

The subject of this observation was a woman, in whom the left kidney was situated deeply on the inside of the psoas muscle. In two labours, through which this woman had passed, a tumour was formed each time on the left side of the pelvis, which excited fixed and increasing pain with each contraction of the uterus; the passage of the head was thereby retarded, but both accouchements were happily accomplished.

The *diagnosis* of a misplaced kidney, forming a pelvic or abdominal tumour, rests on the moderate size and the smooth elastic feel of the tumour, together with the existence of a want of fulness, or a slight hollowing, of the corresponding lumbar region—denoting the absence of the kidney from its usual place. The shape of the tumour, when reniform, of course greatly assists the diagnosis; but in a large majority of such malpositions, the peculiar kidney shape is not preserved.

B. Movable Kidneys.

In addition to the references on p. 501, see :—

Hare—Med. Times and Gaz., 1858, i. p. 7, etc., and 1860, i. p. 30.
Oppolzer—Ibid., 1857, i. 575, and Clin. Europ., 1859, No. 2.
Fritz—Archives Générales, 1859, Aug. and Sept.
Henoch—Klinik d. Unterleibs-Krankh. Berlin, 1858. Bd. iii. p. 367.
Becquet—Archives Générales, 1865, Jan.
Cases—See Lancet, 1862, ii. p. 139; 1863, i. p. 521, ii. p. 363; Med. Times and Gaz., 1857, p. 651; 1858, i. p. 331, ii. p. 36; 1859, ii. p. 426; 1860, i. p. 9; 1864, July 9th; Midland Journ., Jan., 1858; Prag. Vierteljahrschr., Bd. 51.

Vague allusions to mobility of the kidneys are found in the works of the older writers (Mesué and Riolan), but to Rayer belongs the credit of having first pointed out the practical bearing of this condition, and the symptoms and signs by which it may be recognised during life. In this country the subject has been ably illustrated by Dr. Hare, under whose observation no fewer than seven cases have fallen. Mr. Durham has brought together and collated all the instances (10 in number) which, up to that time (1860), had been verified by *post-mortem* examinations. Oppolzer and Henoch, in Germany, have contributed a number of cases; Fritz has analysed all the cases published prior to 1859; and in the present year, Becquet has contributed an essay on the pathogenesis of movable kidneys. The following account is based on an analysis of 51 cases derived from the sources above indicated.

Physical Signs and Symptoms.—The kidneys, in their normal state, are secured in their position by a thick investment of adipose tissue, and a reflection of the peritoneum which passes over their anterior surfaces; but under certain circumstances, one or both kidneys break away from these not very firm attachments, and float loose amid the abdominal viscera—no longer bound except by their blood-vessels and excretory ducts. The degree of mobility and of change of position which the kidney acquires in these cases varies greatly. In the generality of cases, the organ descends, when the patient is standing upright, below the margins of the ribs, and occupies a diagonal position, extending from below upwards and outwards, midway between the costal border and the umbilicus. In this situation it forms an oblong tumour, having the shape and feel of the kidney. It can be pushed in various directions—upwards, or downwards, or laterally—over a space of several square inches. In persons with flaccid bellies, the gland can be actually grasped with the hand; and a sickening, sinking, sensation is experienced when it is compressed; otherwise it is usually painless. When the patient lies horizontally, the displaced kidney can be thrust back again by the hand, into its natural situation in the lumbar region; but it generally resumes its unnatural place when the pressure is withdrawn. The respiratory movements and the posture of the body exercise a marked influence on the position of a movable kidney. Deep inspiration causes it to descend, and deep expiration to ascend; it falls over to the linea alba, or in the opposite direction, as the body is inclined to this or that side. In the slighter cases, half or three-quarters of the length of the organ is palpable through the soft abdominal walls, along the borders of the false ribs: but the displacement is generally more considerable than this; in a case mentioned by Johnson the kidney had drifted below the umbilicus; in another, related by Day, the kidney lay in the iliac fossa, and could be moved hither and thither over a space of three or four inches. When the patient reclines, the displaced organ occupies a higher position than after long standing or walking. Percussion does not yield a dull sound over a movable kidney, but a muffled tympanitic note. When the loins are examined, a flattening or slight hollowing of the renal region, on the side of the displacement, is perceived; and the percussion note is tympanitic—showing that the absent kidney is replaced by intestine. When the organ is thrust back by the hand into its original position, the natural bulging in the loin is restored, and the bowel sound disappears.

The subjective symptoms are somewhat vague. There is usually a feeling, more or less intense, of dragging and weight in the abdomen; sometimes it amounts to no more than a dull uneasiness, chiefly perceived after prolonged standing or exercise. The movements of the displaced organs are distinctly perceived by some patients; in one instance they were mistaken for the supposed movements of a child in the womb. Severe paroxysms, resembling nephritic colic, occurred in some cases. Two of Rayer's cases had neuralgic pains running in the course of the anterior crural and sciatic nerves. Constipation appears always to aggravate the sensations of the patient. Consciousness of the existence of a tumour within the abdomen seems to produce a hypochondriacal state in some individuals.

The secretion of urine, which is always healthy, goes on generally without the least alteration; but in one of Jago's cases there was urgency of micturition when the kidney was pressed towards the pubes, and in one of Henoch's cases the same symptom occasionally occurred on long standing.

The only complications (not clearly accidental) observed were, epigastric pulsation, and congestion and enlargement of the displaced kidney: in three cases (perhaps four) hydronephritic distension of the renal pelvis existed.

The two following examples furnish typical illustrations of movable kidneys:—

CASE I.—Mrs. D., æt. 36, the mother of several children, had been occasionally under Dr. Hare's care for several years. She had suffered from anæmia and oligo-menorrhœa, but got quite well of these. She had afterwards an attack of gastrodynia, when she had also much languor and debility, with weight and sinking sensation at the epigastrium.

In the spring of 1852, Dr. Hare attended her for a slight bronchial attack: when she got better of that she complained of a "beating sensation" down the middle of the abdomen, and also of having at the upper part of it, on each side, "some swellings which on pressure slipped up under the ribs." She had had a sinking sensation at the epigastrium for years, but it was only about twelve months that she had felt, on applying her hand there, a tumour in one (the right?) hypochondrium, and about four weeks, another on the other side. The aortic impulse had been troublesome for five weeks past.

On making an examination of the abdomen (which was rather thin, and of short antero-posterior diameter, while the parietes were also flaccid), the aortic impulse was found to extend from the upper part of the epigastrium to more than an inch below the umbilicus, and it was exceedingly well-marked and strong. The left kidney was situated lower than usual, but readily glided when pressed upon, from under the fingers, deep into the hypochondriac region, while, on the other hand, it might be pushed some distance downwards: the right kidney presented the same phenomena, except that it was much more mobile, and could be detruded downwards so far, that the whole of it could be felt some distance below the costal cartilages, and its form well made out, owing to the thinness of the parietes. (See Fig. 59.)

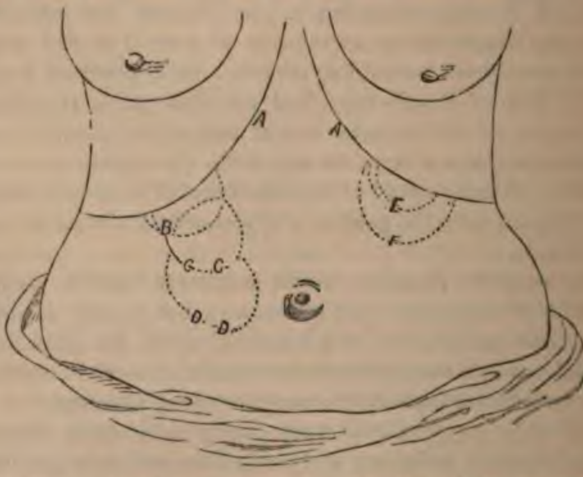

Fig. 59. Diagram showing the varying positions of the kidneys in the case of Mrs. D. A A. Margins of costal cartilages. B. Right kidney; ordinary position when patient is in recumbent position. C C. Ditto, on deep inspiration. D D. Ditto, position to which it can be moved. E F. Left kidney; changes in position of.—(After Hare.)

A belladonna plaster was applied to the abdomen, and some tinct. ferri sesquichlor. and tinct. calumbæ were given. The patient was afterwards seen several times by Dr. Hare, and again very recently: at times she had been free from all renal pain, but lately she had again felt somewhat weaker, and she had had more, both of the abdominal impulse, and of the dragging sensation in the loins, though the pain was by no means so much there as it was a few years ago. On examining the abdomen, the mobility of the kidneys (especially of the left one) appeared to be less than formerly, though that of the right one was still very notable. (*Hare, Med. Times and Gaz.*, 1858, i. p. 86).

CASE II.—A woman, aged forty-three, was admitted into the Charité (Paris) on Dec. 13, 1836.

She was of a phlegmatic temperament and spare habit; but stated that she had never been laid up until the last eight days, during which she had suffered from colicky diarrhœa. The tongue was dirty reddish at the point, white in the centre. There were anorexia and thirst. The abdomen was slightly tender on pressure—no nausea or vomiting.

On examining the abdomen, there was felt on the right side, below the liver, a hard smooth tumour, having the form of the kidney. Its outlines could be plainly traced; it was movable; and could, so to speak, be grasped with the hand, and pushed almost as low as the umbilicus, or thrust under the liver. The tumour was so isolated from the liver, that it could not be confounded with that organ, nor with the gall bladder. In the corresponding lumbar region, a hollow was felt. The position of the left kidney could not be exactly determined. The patient stated that she felt a dragging in the abdomen towards the umbilicus; but she added that this pain encircled the loins: her answers showed that, in addition to the passing derangement of the digestive organs, there was superadded a habitual ailment resembling hypochondria.

There was no pain in the corresponding thigh, nothing unnatural in the urine, and never any interruption to its flow.

The patient had been once pregnant, and had had two miscarriages. By diet and repose the slight gastro-intestinal disturbance speedily subsided; but the tumour and the pains (real or exaggerated) in the abdomen, back, &c., remained.

A diachylon plaster was placed on the belly, and over this a tight bandage, which exercised a strong pressure on the contents of the abdomen. The patient, who the evening before had stated that standing and walking aggravated the abdominal pains, felt relief from the application of the bandage.

She left the hospital Dec. 22, 1836, feeling scarcely any or no pain in the belly. But it was remarked that there was that exaggeration and want of precision in her answers to questions, so common among hypochondriacs. *Rayer*, l. c., iii. p. 784.)

Etiology.—Mobility of the kidneys is much more common in women than in men; and more common on the right side than the left. Of the fifty-one cases which I have been able to collect, forty-six were women and only five men. In forty-six cases, information is given as to the side affected:—

In 28 the right kidney alone was movable.
— 8 the left kidney alone was movable.
— 10 both kidneys were movable.

The age of the patients varied from eighteen to sixty-five years—the general range being between twenty-five and forty, which corresponds roughly to the child-bearing period in women.

In a large number of the cases no clear determining cause could be indicated; in several instances the affection was attributed to the effects of repeated or protracted labours. In one case, related by Henoch, the right kidney became movable after a blow on the right loin: in another, which may be here quoted, from the same author, both kidneys became movable after a fall from a horse.

CASE III.—A military officer fell from his horse some years previously in executing a manœuvre, and descended on his feet, with violent concussion of the entire body, but no immediate evil results followed. About half a year after, he suddenly discovered a swelling in the belly near the navel; a few months later, a second similar swelling was perceived on the left side, which was supposed to be an enlarged spleen. A celebrated surgeon, whom he consulted, judged the tumours, on account of their mobility, to be enlarged mesenteric glands, and recommended the use of Carlsbad waters, but, of course, without any effect.

When examined by Henoch, the following condition was found:—In the upright position of the patient, who was somewhat thin, two slight prominences were visible, one on each side of the navel, and about two inches distant therefrom. These prominences yielded a sonorous sound on percussion, and revealed themselves on palpation as smooth oblong tumours, about the size of goose eggs, with rounded margins. The position and form of the swellings were perfectly symmetrical. Strong pressure upon them did not exactly excite pain, but caused a sensation resembling, as the patient said, that produced by squeezing the testicles. By continuous pressure upwards and backwards, both tumours could be easily thrust into the lumbar regions; but they resumed their old position when the pressure was withdrawn. The tumours also disappeared when the patient reclined on the back, but on resumption of the upright posture they became again perceptible in a short time. When the patient lay quietly on his back, he could render the vanished tumours again palpable, by strongly pressing with his thumbs in the lumbar region; the tumours then sprang forward with elasticity. The lumbar regions, when the patient stood upright, were somewhat sunk-in. There were no urinary symptoms. The only inconvenience which the patient experienced, consisted in an unpleasant dragging sensation in the standing position, which evidently depended on the weight of the depressed organs. He also complained of diminishing strength in the lower limbs; this was not, however, due to the mobility of the kidneys, but, as was proved later, to a commencing tabes dorsalis. An abdominal bandage did not in this case produce any good effect. (*Henoch*, l. c., Bd. iii. p. 368.

The disproportionate frequency of mobility of the kidney in the female sex, and on the right side of the body, indicates that tight lacing is probably not without effect in dislodging the kidney and rendering it movable. Cruveilhier remarks:—"I have often observed, in women who wore tight stays, the right kidney to lie sometimes in the right iliac fossa, sometimes in front of the sacro-iliac synchondrosis, sometimes even in front of the vertebral column, at the level of the adherent border of the mesentery, in the substance of which it was placed. The kidney, thus accidentally displaced, enjoys a certain mobility. This displacement of the kidney arises, when the pressure, exercised on the liver by the stays, dislodges the right kidney from the kind of niche which it occupies on the under surface of this organ.

"If the left kidney is not so frequently displaced as the right, that is owing to the fact that the left hypochondrium, occupied by the spleen and the great end of the stomach, bears the pressure of the stays with much more impunity than the right." (*Cruveilhier*, *Descriptive Anat.*, vol. iii.)

Rapid emaciation in obese persons, and the removal of the capsule of adipose tissue which naturally invests the kidney, seem, in some instances, to have favoured or determined the mobility of the organ. Oppolzer states, that in the cases which he had an opportunity of examining the patients dying of some other disease, there had always been observable, a deficiency of the cushion of fat about the kidney. In a case dissected by Mr. J. Adams, "the only peculiarity remarkable was, that the kidney appeared bound down in its situation more loosely than usual, and the old lady, from having been very fat, had become somewhat thinner, and her integuments appeared very lax throughout." (*Med. Times and Gaz.*, 1857, i., p. 651.)

Rayer mentions a case in which it appeared probable, that the kidney was dragged down, or at least left free to descend from its own weight, in consequence of displacement of the peritoneum, from a hernia of the cæcum.

The coincidence of hydronephrosis in so many as three, if not four, cases, can scarcely be regarded as accidental. The pressure of the distended pelvis, and the increased weight of the organ, had probably an important influence in dragging the kidney from its natural situation.

Becquet has proposed a somewhat novel theory for the production of movable kidneys in women. In the cases encountered by him, there was a striking coincidence of time, between the displacement of the kidney, and the menstrual period; and he was led to believe, that the kidney became congested and tumefied at these periods, and that displacement was the consequence of its increased volume and weight. He thus explains himself:—"On the breaking forth of the menstrual flux, the kidneys are associated in the congestion of the generative organs, and become swelled. This fact, less rare doubtless than is usually supposed, perhaps even physiological, does it not explain the renal pain so often felt at the menstrual periods, especially in women who are subject to dysmenorrhagia?"

"Thus swelled and rendered heavier, the kidney, and especially the right kidney, strains the feeble attachments which retain it, and tends to start out of its place. Soon the congestion subsides, and the organ returns to its original position; a second congestion displaces it further; and a third further still: the kidney, becoming each time heavier from the incompleteness of the resolution, comes to occupy a lower position; and thus gradually, and at length, but not without suffering, breaks loose, and floats in the abdominal cavity." (Arch. Gén. 1865, i. p. 21.)

But although, in a majority of the cases, mobility of the kidney appears to have been acquired from some accident or circumstance arising in the course of life, there are instances in which a congenital anomaly in the anatomical connections of the gland has evidently operated to favour its production. In two cases,* the peritoneum was found reflected over the posterior, as well as the anterior, surface of the kidney, so as to enclose it within a fold of peritoneum, or meso-nephron, which permitted the organ very considerable motion in the abdomen. Mr. Durham examined the body of a woman, aged 34, in which the left kidney was very movable. He found the descending colon much nearer the middle line than usual, and, instead of forming the sigmoid flexure in the left iliac fossa, it turned across the lumbar vertebræ, and passed down into the pelvis on the right side of the sacrum. It was manifest that the mobility of the kidney in this instance depended, in great measure, on the abnormal arrangement of the peritoneum, necessarily associated with the malposition of the colon. So far, therefore, it must be regarded as congenital. When traced from the side of the vertebral column, the peritoneum, instead of passing over the anterior surface of the kidney, only just touched the lower part of its inner border, and then, having formed the descending meso-colon, again touched its outer border. The lesser sac of the peritoneum also, instead of being confined to its ordinary limits, passed so far to the left as to cover the posterior surface of the spleen, and so far downwards as to touch, and be reflected from, the upper border of the kidney. Thus there was no distinct meso-nephron; but the kidney, instead of being supported and kept down by a single layer of peritoneum, was left free to move between and beneath three diverging layers. Upon dissection it was further found, that there was scarcely any fat in the lumbar region, but a quantity of very loose areolar tissue. (Guy's Hosp. Rep. 1860.)

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* Dr. Priestley's case—Med. Times and Gaz., 1857, i. 263; and Girard's case—cited by Rayer, l. c., iii. p. 793.

The *diagnosis* of movable kidneys is chiefly important, from the risk of confounding an ailment which is comparatively trifling, with some graver disease. In a considerable number of instances, the affection was long mistaken by the patient and his medical attendants for a tumour within the abdomen; and the patient was subjected, in addition to the alarm which such a notion necessarily engendered, to heroic and exhausting or troublesome plans of treatment. The diagnosis is indeed generally easy; and the errors committed have arisen, from the possibility of this condition not having been present to the mind of the practitioner, rather than from the inherent obscurity of the case.

A movable tumour having the size and shape of the kidney, or approaching thereto, is found on either side of the abdomen, generally in the hypochondriac region; it can be pushed into the lumbar space, and again out of it, at will, by the thumb and fingers. When the corresponding loin is examined, the absence of the kidney from its usual place is rendered evident, by the flattening or hollowing of the part, and by the tympanitic note yielded on percussion. It is only in very obese individuals, and in cases where the displacement and mobility are slight, that any difficulty can arise. It must be remembered, that the displaced organ sometimes contracts adhesions in its new position, and thereby loses, partly or wholly, its freedom of movement.

Treatment.—Persons with movable kidneys do not, usually, suffer serious inconvenience therefrom; and the affection generally persists without much change for an indefinite period. The dragging sensations in the abdomen were, in most of the cases, relieved by wearing a tight elastic bandage round the loins; in other cases no benefit was experienced from this treatment. If there be anæmia, or other disorder of health, the removal of this by appropriate remedies is of course to be attempted. Restoration of the tone of the abdominal muscles, which, in most cases, are relaxed and flaccid, is probably the most effective means of reducing to a minimum the inconveniences which attend on mobility of the kidneys. To this end, ferruginous and other tonics, and shower baths, with avoidance of fatiguing exercise, seem to be the means best adapted. A curious case is recorded by Dr. Hare, in which the mobility of the kidneys was markedly diminished after two pregnancies; the steady pressure of the gravid uterus having apparently acted as a mechanical support to the dislodged organs.

In cases, like those described by Becquet, in which the displacement is accompanied with violent paroxysmal pains in the loins, and coincidence of the menstrual flux, complete repose should be prescribed during the attacks, and hot poultices, or even leeches, applied to the seat of pain. In the intervals of the paroxysms Becquet recommends the employment of hydrotherapeutic means.

The regulation of the bowels is a point to be carefully attended to. Accumulation of fæcal matter in the large intestines invariably aggravates the inconveniences of movable kidneys. Tight lacing and all violent modes of exercise (equitation, dancing) should of course be strictly forbidden.

II.—ANOMALIES OF FORM.

Deviations from the normal shape of the kidneys may exist congenitally, or be produced in after life by the pressure of tumours or of enlargements of the neighbouring organs. Some of these malformations have been already noticed, in treating of fixed misplacements of the kidney.

The lobulated character of the gland, which is natural to it in the fœtal state, sometimes persists more or less throughout life. Sometimes one kidney is twice or thrice as large as its fellow, although both may be perfectly healthy—an anomaly probably due to deficient development of one renal artery.

The pelvis of the kidney, and the ureter, sometimes present curious anomalies. Mr. H. Thompson encountered a kidney with two pelves, which united into a single ureter about an inch below their necks (Path. Soc. Trans. vi. 267). In a case recorded by Mr. Wood (Ibid. vii. 261), the left kidney had two ureters, which continued distinct until within an inch of the bladder. The right kidney of the same patient had, in addition to a ureter which entered the bladder at the usual place, an aberrant ureter, connected with a dilatation (partial hydronephrosis) at the upper extremity of the kidney; this aberrant duct was as thick as a goose-quill, sacculated, and opened into the bladder close to the exit of the urethra.

Horse-shoe kidney.—The most common deviation from the normal shape of the kidney, consists in the fusion of the two organs into one, by an intermediate transverse portion, or isthmus, which connects their lower ends across the spine, so as to form a crescent or horse-shoe. Fig. 60 represents a specimen which I removed some years ago, from the body of a patient who died of phthisis in the Royal Infirmary. The two halves of a horse-shoe kidney are usually complete and perfect in themselves, and possess each a separate pelvis and ureter. The transverse portion is generally composed of proper secreting structure; but sometimes it consists merely of condensed fibrous tissue. The concavity of the crescent is always directed upwards; and the ureters generally descend in front of the transverse portion—but sometimes, according to Wilks, behind it. In a drawing possessed by my colleague, Dr. Renaud, the two ureters of a horse-shoe kidney are seen to cross each other on their way to the bladder. The arterial supply of a horse-shoe kidney always presents some departure from the ordinary distribution.

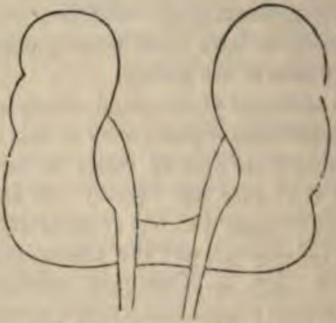

FIG. 60. Horse-shoe kidney.

This deformity does not occasion any derangement in the secretion of urine, provided the organ remain healthy. Rayer reminds practical men, that in thin persons, with flaccid bellies, the transverse portion of a horse-shoe kidney may give the feel of a morbid growth in the abdomen; and that suppuration and dilatation of the pelvis of such a kidney may occasion a tumour, which, from its central position near the spine, would lead an observer away from the idea of pyonephrosis, unless the possibility of this deformity were borne in mind.

III.—ANOMALIES OF NUMBER.

Rayer cites a number of instances in which there existed one or two supernumerary kidneys, each with its separate excretory duct. The same author cites examples of still-born infants, presenting a complete absence of both kidneys, together with the ureters and bladder. In acephalous monsters this abnormity appears to be not uncommon.

Solitary kidney.—The absence of one kidney has been repeatedly observed in the bodies of persons, who presented no derangement of the urinary function during life. The existing organ, in such cases, is always hypertrophied; and so long as it remains healthy, the secretion of urine is carried on without appreciable defect. But if the solitary kidney become inflamed, or its excretory duct obstructed by the impaction of a calculus, or the pressure of a tumour, alarming symptoms speedily make their appearance, accompanied with partial or total suppression of urine, ending in fatal uræmia.

Of fourteen cases of solitary kidney collated by Mosler,[*] death was caused in nine, by the impaction of a calculus in the ureter. In the remainder, death resulted from various causes—inflammation of the kidney, pressure of a cancerous tumour on the ureter, impeded flow of urine from congenital phymosis. Three-fourths of the cases were males. The two sides were affected with almost equal frequency. One was a male infant seven days old; another a girl of fifteen years; most of the cases had attained middle age—the oldest was sixty. In twelve of the cases, the defect was congenital; in two, it had been acquired later in life through destruction of the opposite organ. The renal vessels and the ureter of the defective side were always absent in the congenital cases. The corresponding supra-renal capsule was likewise generally wanting when the defect was congenital.[†]

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[*] Archiv d. Heilk. 1863, p. 290.

[†] In addition to the cases collected by Rayer and Mosler, the following examples of solitary kidney may be referred to:—Garrod—Lancet, 1863, ii. p. 724; Ogle—Path. Soc. Trans. 1851-2, p. 382; Sydney Jones—Ibid., vol. viii. p. 279; Murchison—Ibid., vol. x. p. 190; Hillier—Ibid., vol. xv. p. 46.

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